RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

PUBLISHED BY THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

Vol. 38 MARCH 1942 No. 3

Some Practical Considerations Regarding the Employment of Various Qualities of Roentgen Rays in Therapy[1]

EDITH H. QUIMBY, Sc.D.

Memorial Hospital, New York City

FROM THE PHYSICAL point of view, the essential problem in radiation therapy is to deliver an adequate amount of radiation to the cells it is desired to affect, without at the same time over-irradiating normal structures. What constitutes an adequate amount of radiation in any particular case must be determined by the radiologist, on the basis of the nature and location of the tumor and the personal characteristics of the individual patient. While in planning treatment his first consideration should be the amount of radiation he would like to deliver to the tumor, he will also pay attention to skin doses and to probable systemic effects. The rate at which the radiation is to be administered, including the intensity of the beam, the magnitude of each fractional dose, and the frequency of treatment, is of primary importance.

In determining the preferable procedure for irradiation in any individual case, the radiologist must have recourse to physical data regarding the effects of variations in the physical factors on the surface and depth doses delivered. The author has been requested to discuss the problem of the delivery of a predetermined adequate dose to any specified lesion, with the resources at the command of the average radiation therapist.

From the physical point of view, this can involve only the consideration of the relative merits of various qualities of radiation in regard to doses delivered to various points. Such data can have little or no bearing on the time factor, except to determine whether the desired rate of irradiation is practicable. Accordingly the present paper is concerned with setting forth certain physical data in convenient form for practical use, and in discussing some applications of these data. Little new material is included.

At the present time it is unfortunately true that considerable carelessness exists in the specification of radiation dosage. Too often the actual tumor dose is overlooked; the amount of radiation delivered "in air," or "to the skin, including backscatter," is the complete statement of the treatment. Thus two patients, each of whom was exposed to 1,000 roentgens measured in air would be said to have received the same dose. Yet in one case the lesion might have been 2 cm. below the skin in a 25-sq. cm. field, and in the other, 7 cm. below, in a 100-sq. cm. field. Actually, the first lesion will have received from 50 to 150 per cent more than the second, depending on the quality of the radiation and the focal-skin distance.

[1] Presented before the Radiological Society of North America, at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

Frequently even the qualifying expression "in air" or "on the skin" is omitted, the difference in actual tissue dose for the same number of roentgens specified in the two ways appearing not to be appreciated. There can, of course, be no single factor representing the relation between the two. An air dose of 1,000 r may mean a skin dose of as much as 1,500 r for moderately hard radiation and a large field, or as little as 1,050 r for quite hard (or very soft) radiation and a small field, according to the variations in back-scatter. Doses at any depth within the tissue differ correspondingly.

To specify only the air dose, or the skin dose, may give a false sense of adequacy. The statement that 2,500 r was delivered to a 100-sq. cm. field sounds like a considerable amount, yet, as will be seen from data to be presented, for 200-kv. radiation with a filter of 2 mm. copper and a distance of 80 cm., the tissue dose at a depth of 12 cm. is only 800 r, and for 200 kv. with 0.5 mm. copper and 50 cm. distance, only 540 r. It is probable that neither of these doses would be considered adequate for the treatment of carcinoma.

On the other hand, by focussing the attention on the tumor dose rather than on air or skin dose, the radiologist may avoid serious damage due to over-irradiation. Much has been said about the desirability of treating each patient as an individual, and not using a routine technic for all cases of a certain type, but the custom persists in many institutions of giving a "standard pelvic cycle," a "standard postoperative cycle after radical mastectomy," etc. More than one radiologist, having established a routine pelvic cycle which seems satisfactory in the average case, has found himself confronted with disastrous bladder and intestinal damage after employing the same cycle on a woman weighing 100 pounds or less. If he had considered that the dose to the intestines in a woman 16 cm. in anterior-posterior diameter is of the order of twice as great as in a woman 24 cm. in diameter, he might not have fallen into this difficulty.

No single set of physical factors—voltage, filter, and distance—is satisfactory for all radiation therapy. For superficial lesions, the use of very penetrating beams may entail heavy irradiation of underlying vital structures, which could be avoided by the employment of softer rays. On the other hand, for deep-seated lesions, it is necessary to use the most penetrating radiation available. Thus the therapist needs to have at his command a range of qualities, and to know the limitations of each one.

The qualities of radiation to be considered in the present discussion will be limited to those which should be within the reach of every therapist, that is between 100 and 200 kv. This does not imply that there is no value in lower or higher voltages; the limits have been set simply by the generally available equipment. The radiologist who is so fortunate as to possess facilities for contact therapy or supervoltage installations will find dosage data in the literature, to supplement those here presented.

In the so-called high-voltage region, 200 kv. is usually employed, with filtration of 0.5, 1.0, or 2.0 mm. of copper or the corresponding composite filters, and with distances from 50 to 80 cm. In the low-voltage range there is no such general agreement; a very large number of voltage-filter-distance combinations are employed in various x-ray departments. Among these, it is possible to select groups of factors which look quite different, yet which actually give essentially the same tissue doses. For instance, 100 kv. with 2 mm. aluminum at 30 cm. distance, 120 kv. with 1 mm. aluminum at 25 cm., and 120 kv. with 4 mm. aluminum at 15 cm. give depth dose curves which can hardly be distinguished from one another.

Since it is difficult to present useful tables or charts containing many sets of physical factors, it seems desirable from a practical point of view to select a reasonable group and limit discussion to these. Accordingly, six voltage - filter - distance combinations have been chosen as follows:

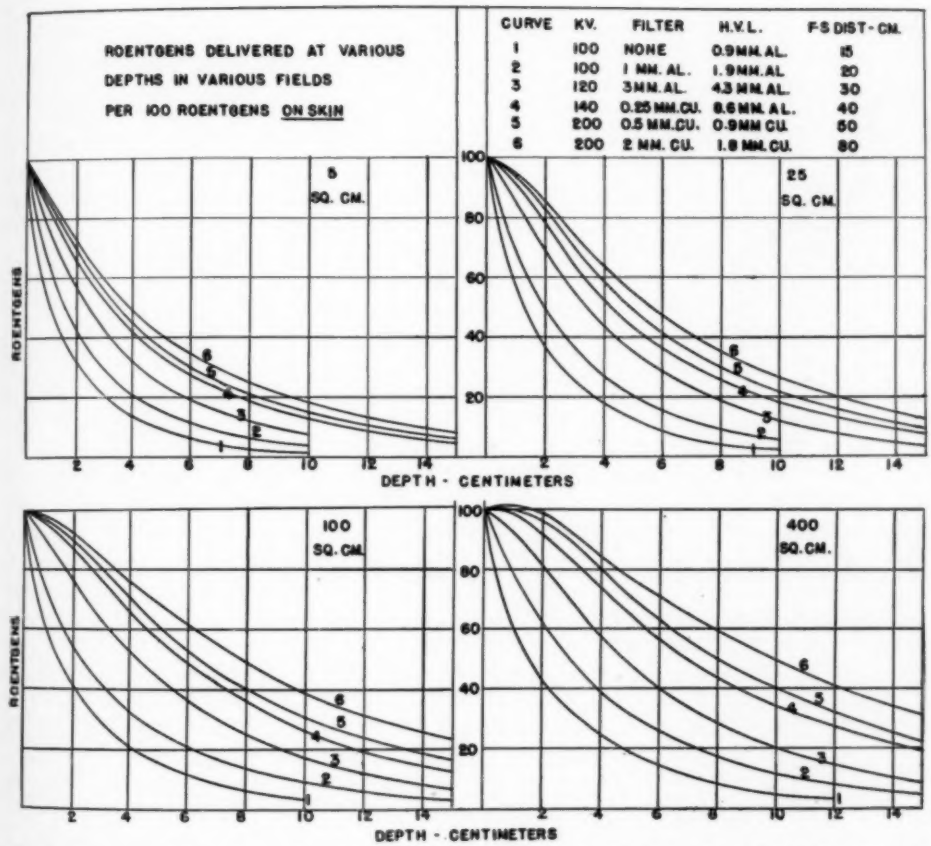


Fig. 1. Roentgens delivered at various depths per 100 roentgens *on the skin*, for various fields and qualities of radiation. Roentgen dose scale in middle of chart.

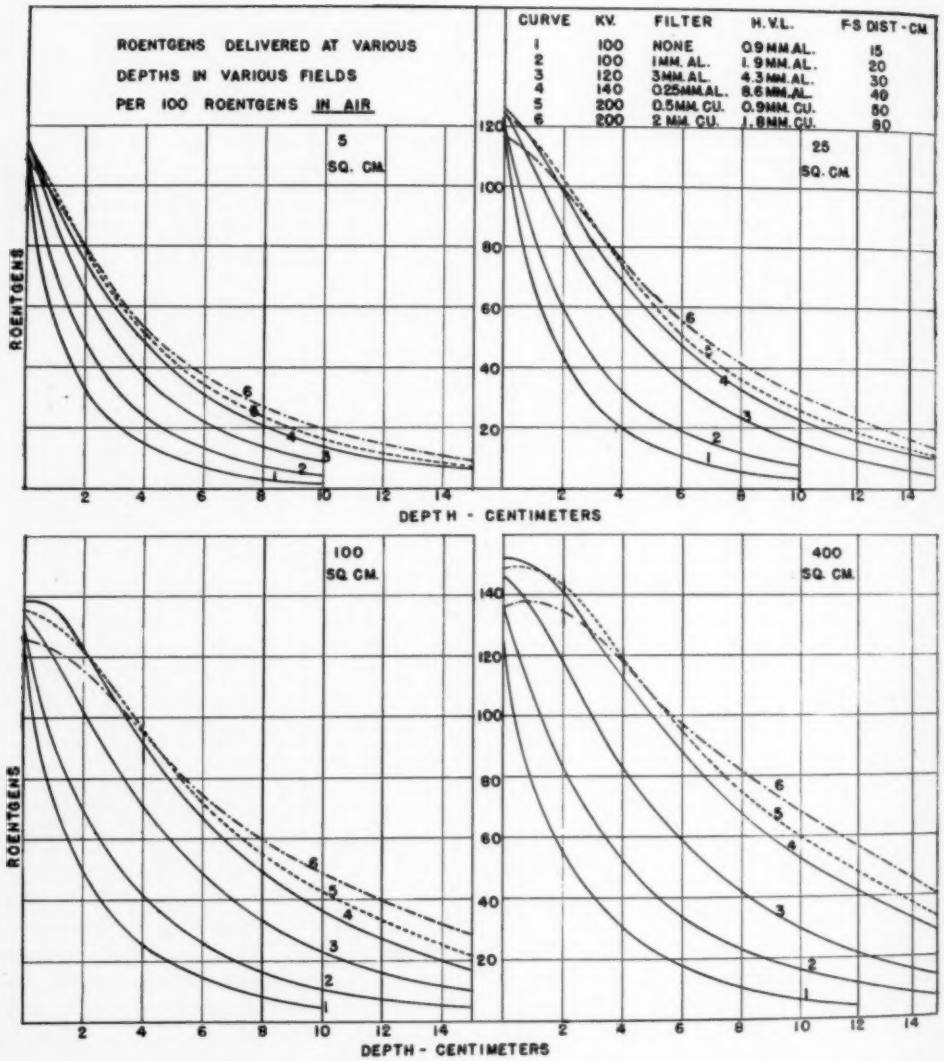


Fig. 2. Roentgens delivered at various depths per 100 roentgens *in air*, for various fields and qualities of radiation. Roentgen dose scale in middle of chart.

Kilovolts	Filter	Half-Value Layer	Focal-Skin Distance	r/min. (air) (Approximate)*
100	0	0.9 mm. Al	15 cm.	400
100	1 mm. Al	1.9 mm. Al	20 cm.	150
120	3 mm. Al	4.3 mm. Al	30 cm.	40
140	0.25 mm. Cu	8.6 mm. Al	40 cm.	15
200	0.5 mm. Cu	0.9 mm. Cu	50 cm.	45
200	2.0 mm. Cu	1.8 mm. Cu	80 cm.	8

* These values are based on averages of calibrations made on low-voltage machines operating at 4–5 ma., and on high-voltage machines operating at 20–25 ma.

It is not implied that this is the only desirable set of qualities or, necessarily, the best. They are, however, adequate within the voltage range under consideration. They have been chosen to cover the region in nearly equal steps for depth doses, and to be such that the output of radiation is in no case too small to be practical.

For each of these qualities, depth dose curves have been plotted for four fields, 5, 25, 100, and 400 sq. cm. Since at the present time radiologists are divided between use of air dose and of skin dose as starting point, separate charts have been made for the two.[2] In Figure 1 are the curves for depth dose in percentage of skin dose, that is, roentgens delivered at any depth *per 100 r on the skin*. In Figure 2 are the corresponding curves for depth dose in percentage of air dose—roentgens delivered at any depth *per 100 r in air*. In this latter case, because of the decreased back-scatter for the harder radiation, the initial points of the 200-kv. curves lie below those for 120 and 140 kv.

These curves are all for irradiation with open ports, the field sizes being defined by lead diaphragms, and with no cones to limit the beam. The use of cones with closed ends alters these depth doses somewhat, because of the scattering of radiation in the material in contact with the skin of the patient. For very soft radiation, depth doses with such cones may be somewhat higher than without; for harder rays, depth doses with cones are in general somewhat less; the amount of the variation depends on the material and thickness of the cone cover. For covers of organic material, new sets of curves can be plotted from those of Figures 1 and 2 in the following manner:

Determine the thickness of the cover, and the depth dose at a depth corresponding to this thickness. This becomes the new 100 per cent; doses for all other depths must be recalculated from this reference point. For instance, assume a cone cover of 1/4 inch bakelite on a 100-sq. cm. cone, to be used for 200 kv., 0.5 mm. Cu, 50 cm. One-fourth inch is approximately 6 mm. In Figure 1 the dose at this depth on the appropriate curve is 98 per cent. Five cm. deeper, or at 5.6 cm. on the curve, the dose is 56 per cent. For the new curve, in which 98 per cent becomes 100, 56 becomes 56/98 or 57 per cent. This is the depth dose for the point 5 cm. below the skin under the cone and is definitely less than the point 5 cm. below the uncovered skin, which, according to the curve, is 61 per cent.[3]

In addition to the relative depth doses as determined from Figures 1 and 2, it is desirable to know something about the absolute rate of administration of the radiation, from the economic as well as the clinical point of view. If a quality of radiation which appears desirable can only be delivered so slowly that it is doubtful whether the patient can be kept quiet and comfortable for the treatment, for instance, it is well to see whether another quality cannot be employed without serious sacrifice of tissue dose. In Figure 3 are given

[2] The data used have all been obtained at the Memorial Hospital, using the extrapolation ionization chamber. Much of the material has been published elsewhere, and it has been collected in *Technical Bulletin No. 1* of the Standardization Committee of the Radiological Society of North America (1).

[3] Considerable data for depth doses with various cone covers will be found in the *Technical Bulletin No. 1* already referred to.

average values of the actual r per minute delivered at various depths by the six qualities of radiation at the distances used in Figures 1 and 2, for a 100-sq. cm. field, by the x-ray equipment in general use at the present time. In the individual case, of course, some of these curves may be higher or lower than in the figure, but these give a fair comparison.

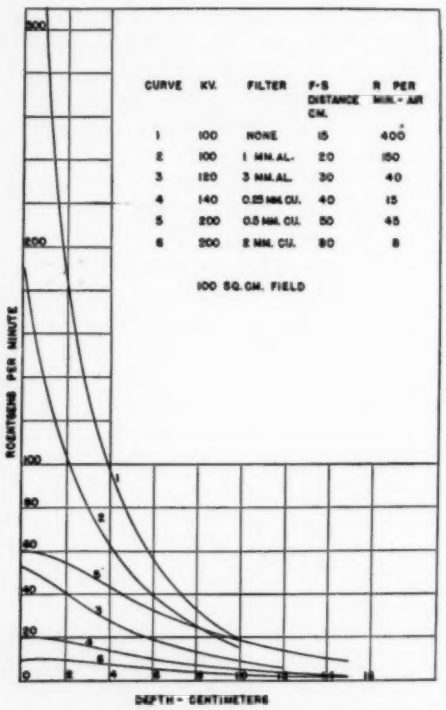


Fig. 3. Roentgens per minute delivered on skin and at various depths by x-rays of various qualities. Values of intensities are averages of calibrations in a number of institutions.

Besides the four fields given in the charts, others will be employed by all radiologists. For doses with such fields, interpolation may give a satisfactory value; for instance, doses in a 50-sq. cm. field are about half way between those for 25 and those for 100 sq. cm. It is, however, more satisfactory to plot the data for a particular quality of radiation in the form shown in Figure 4. Such a set of curves can be prepared for any particular quality of radiation and distance, from the data of Figure 1 or Figure 2, and may well be posted near the x-ray machine to which they apply. For the very soft radiations, large fields are seldom employed, and depths of more than 5 cm. are of little significance. In this case, a more detailed plot of the data for small fields and shallow depths is desirable; such a one is given in Figure 5, for two such qualities. For other soft radiations, the variation from field to field for any particular depth is the same as for those plotted, so that similar charts can be made for them as desired.

The radiologist may also employ other distances than those assigned to the various qualities in Figures 1 and 2. If he has ascertained by calibration that the inverse-square law holds between the distance he intends to employ and the one used in the chart, he can calculate the change in depth dose in accordance with this law. It is convenient, however, to have a table for this purpose, such as Table I.

For convenience, all depth doses have been referred to those at 50 cm. distance; this, however, is immaterial. The table can be employed for any shift in distance within the limits tabulated, provided only that equal doses are delivered on the skin in the two cases. For example, in order to find out what depth dose would be delivered by the 200-kv. radiation with 0.5 mm. copper filter and a 400-sq. cm. field at 70 cm. instead of 50, to a lesion 10 cm. deep: According to the table, the 10-cm. depth dose at 70 cm. is in the ratio of 110.3/100 to that at 50 cm. From Figure 1, for a 400-sq. cm. field the 10-cm. depth dose is 40 per cent of the skin dose. Hence for 70 cm. it would be $110.3/100 \times 40 = 44$ per cent of the skin dose.

Again, suppose it was desired to use 140-kv. radiation with 0.25 mm. copper at 20 cm. instead of 40, for a lesion 3 cm. deep in a 25-sq. cm. field, in order to administer the treatment as rapidly as possible. For the same skin dose, the time would be reduced to one-fourth what it was at 40 cm. ($40^2/20^2 = 4$). The depth dose at 3 cm. for the shorter distance is

TABLE I: FACTORS FOR DETERMINING DEPTH DOSES AT VARIOUS FOCUS-SKIN DISTANCES, IN TERMS OF EACH DEPTH DOSE FOR 50 CM. DISTANCE AS 100 PER CENT
(Skin Dose 100 Per Cent for Every Distance)

	Focus-Skin Distance in Centimeters									
	15	20	25	30	40	50	60	70	80	100
Depth-Cm.	Percentage of Depth Dose at 50 Cm.									
0	100	100	100	100	100	100	100	100	100	100
1	91.5	94.5	96.2	97.5	99.5	100	100.8	101.1	101.4	102.0
2	84.1	89.4	92.8	95.0	98.3	100	101.2	102.2	103.2	104.0
3	78.0	85.0	89.5	92.6	97.4	100	102.0	103.3	104.2	106.0
4	72.8	81.0	86.8	90.6	96.6	100	102.5	104.6	105.8	107.8
5	68.0	77.5	84.0	88.9	95.8	100	103.1	105.7	107.0	109.4
6	64.0	74.4	81.5	87.0	95.2	100	103.8	106.4	108.5	111.5
7	60.5	71.2	79.8	85.5	94.4	100	104.1	107.4	109.8	113.6
8	57.3	68.6	77.2	83.7	93.6	100	104.9	108.4	111.0	115.5
9	54.5	66.2	75.2	82.2	93.0	100	105.5	109.3	112.2	117.0
10	51.9	64.0	73.5	81.0	92.4	100	106.0	110.3	113.9	119.2
11	49.6	61.9	71.8	79.5	91.8	100	106.4	111.3	115.0	120.9
12	47.5	60.1	70.2	78.5	91.0	100	106.9	112.2	116.3	123.0
13	45.6	58.2	68.8	77.5	90.7	100	107.1	113.0	117.4	124.2
14	43.8	56.6	67.2	76.0	90.4	100	107.7	113.9	118.8	126.0
15	42.4	55.2	66.0	75.2	89.5	100	108.1	114.9	120.0	127.2

TABLE II: NUMBERS OF ROENTGENS DELIVERED AT VARIOUS LEVELS WHEN EQUAL DOSES ARE ADMINISTERED AT PARTICULAR DEPTHS BY DIFFERENT QUALITIES OF RADIATION, TOGETHER WITH COMPARISON OF TIMES REQUIRED FOR DELIVERY OF THE EQUAL DOSES

Kilovolts	100	100	120	140	200	200
Filter	None	1 mm. Al	3 mm. Al	0.25 mm. Cu	0.5 mm. Cu	2.0 mm. Cu
Focus-Skin Distance	15 cm.	20 cm.	30 cm.	40 cm.	50 cm.	80 cm.
	100 Roentgens at 1 Cm. Depth—5 Sq. Cm. Field					
Surface	200	160	130	122	118	This quality would
5 cm. Depth	18	25	32	40	41	not be consid-
10 cm. Depth	3	8	10	15	17	ered in this case
Relative Times	1.0	2.1	7.7	16.0	5.0	
	100 Roentgens at 3 Cm. Depth—25 Sq. Cm. Field					
Surface	410	280	185	156	143	135
10 cm. Depth	11	17	22	28	30	35
Relative Times	0.24	0.42	1.0	2.2	0.7	3.9
	100 Roentgens at 5 Cm. Depth—50 Sq. Cm. Field					
Surface	These qualities would		250	200	180	165
10 cm. Depth	not be considered in		36	44	47	53
Relative Times	this case		0.5	1.0	0.3	1.7
	100 Roentgens at 12 Cm. Depth—100 Sq. Cm. Field					
Surface	These qualities would not be consid-			530	430	325
Relative Times	ered in this case			0.8	0.2	1.0

85/97.4 of that for the longer. From Figure 1, at 40 cm. with a 25-sq. cm. field it is 64 per cent; hence at 20 cm. it would be $64 \times 85/97.4 = 56$ per cent of the skin dose. This is a considerable loss in dose; it might not be considered a desirable change in treatment plan.

In selecting the quality of radiation to be employed for any particular lesion, as has been stated above, the radiologist will take into account not only the dose at that definite depth, but also the doses to overlying and underlying structures. In Table II, data from Figures 1 and 2 have been arranged to permit of rapid comparison in some special cases. Here, when equal doses are delivered at certain levels, the simultaneous doses at other levels are tabulated. The application of this information to some special problems may be considered.

1. A small superficial lesion at the outer canthus of the eye, greatest possible depth less than 1 cm., skin damage unimportant, but radiation to the

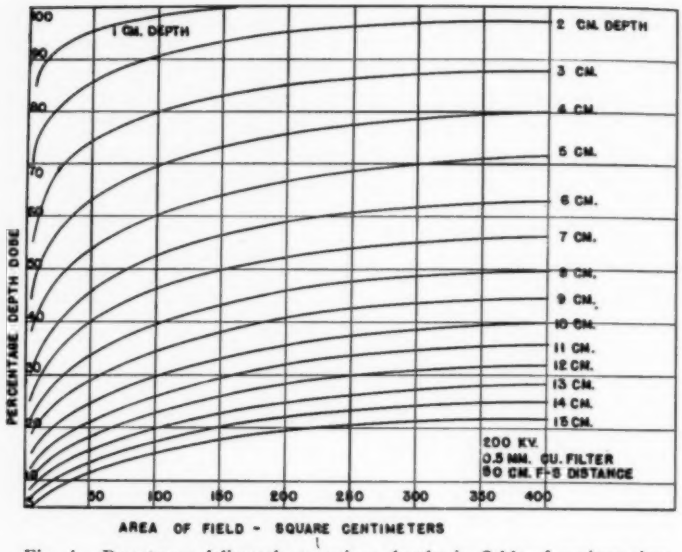


Fig. 4. Roentgens delivered at various depths in fields of various sizes, per 100 roentgens on the skin: 200 kv., 0.5 mm. Cu filter, 50 cm. F-S. distance.

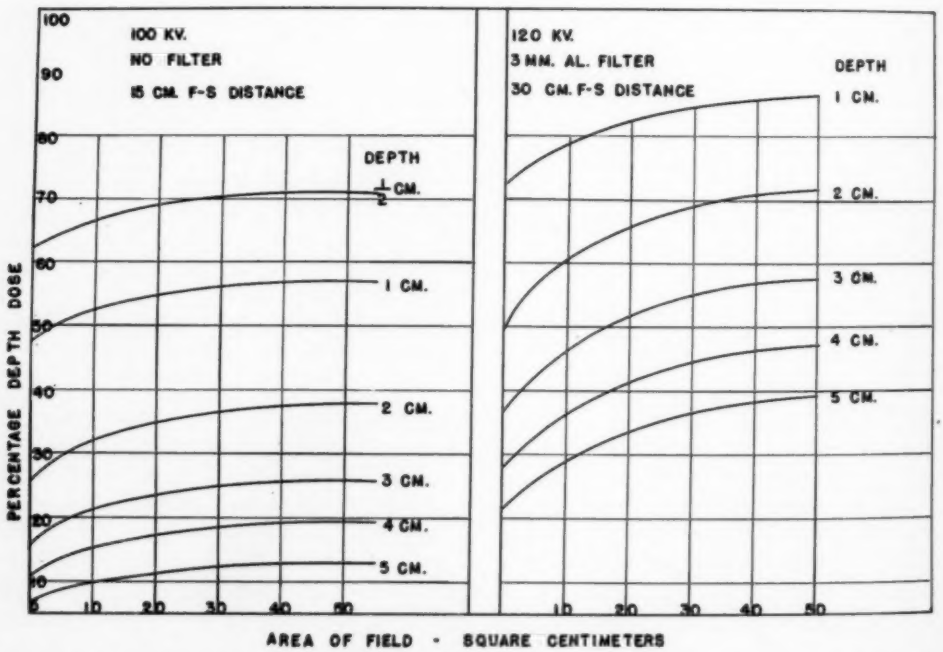


Fig. 5. Roentgens delivered at various depths in fields of various sizes, per 100 roentgens on the skin. I. 100 kv., no filter, 15 cm. F-S. distance. II. 120 kv., 3 mm. Al filter, 30 cm. F-S distance.

lenses of both eyes to be kept to a minimum. In the first part of the table it is seen that for equal doses at 1 cm. depth, 100-kv. unfiltered radiation at 15 cm. delivers only about half as much to the adjacent eye (approximately 5 cm.) and one-third to the other eye (10 cm.) as 120 kv. with 3 mm. aluminum and 30 cm. distance, while the difference with any harder radiation is still greater.

2. A local recurrence in the chest wall after radical mastectomy, greatest depth 3 cm. It is desired to avoid skin damage and also lung damage; the field is to be 25 sq. cm. From the point of view of lung damage, the softest radiation would, of course, be best, but with this it appears that for equal tumor doses, the skin dose would be more than twice as much as for an intermediate quality—120 kv. with 3 mm. aluminum. The skin dose for this latter quality is 185/143 or 30 per cent greater than for 200 kv. with 0.5 mm. copper, when equal tumor doses are administered, while at a depth of 10 cm. in the lung the dose is 22/30, or about 30 per cent less.

3. In an average-sized woman, could adequate irradiation be delivered to a pelvic growth by so-called intermediate therapy? For equal doses at 12 cm. depth in a 100-sq. cm. field, the 140-kv. beam delivers 23 per cent more to the skin than 200 kv. with 0.5 mm. copper at 50 cm., and 63 per cent more than 200 kv. with 2 mm. copper at 80 cm. Furthermore, the intermediate voltage radiation is more effective than the higher in producing skin damage.

From these examples it is evident that it is seldom that one set of physical factors is obviously better than all others. The advantages and disadvantages of the various possibilities must be weighed, and final decision rest on this.

It is sometimes argued that it is difficult or impossible to know the actual depth of a lesion, and hence impossible to determine the tissue dose accurately, and that, since the air or skin dose can be known accurately, it is better to specify treatments by this means. This, of course, begs the question referred to earlier, of the great differences in tissue doses at various depths within a particular beam. It is, furthermore, completely invalid in practice. Obviously, careful measurement of the patient is necessary in order to know the depth with reasonable accuracy, but this is not an impossibility, and the fact that it requires a little time should not be accepted as an excuse for not doing it. It is true that an error of 1 cm. in the estimated depth may introduce an error as large as 20 per cent in the dose *if a single beam is employed.* (Even this is better than the complete uncertainty resulting from no attempt at statement of tumor dose.) In general, however, for deep-seated lesions, some form of cross-fire is used, in which case a shift of 1 cm. seldom introduces an error as large as 10 per cent. This is illustrated by Figure 6,

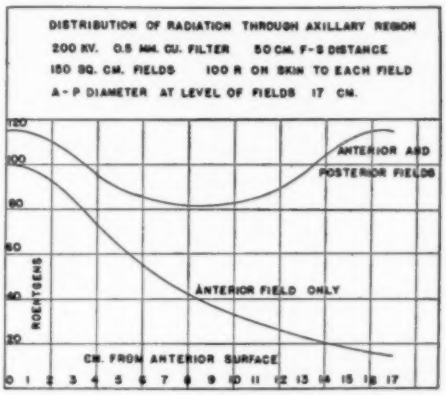


Fig. 6. Effect of cross-fire on amount of radiation delivered throughout axillary region: 200 kv., 0.5 mm. Cu filter, 50 cm. F-S. distance, 150 sq. cm. field.

showing the difference in the amount of radiation delivered throughout an axillary region by a single beam of 200-kv. x-rays and by two beams of the same quality cross-firing through opposite portals. The lower curve indicates the number of roentgens at different depths per 100 r on the anterior skin field. Within the possible node-bearing region, say from 3 cm. depth to 9 cm., there is a very large variation in the possible dose, and an error of 1 cm. in determining the position of a node may mean an error of about 15 per cent in the calculated dose. The upper curve indicates the number of roentgens at the same positions for 100 r to an anterior and 100 r to an opposite posterior skin field. The exit doses bring the total skin doses to 115 r each; it is evident that throughout the possible node-bearing region the differences in dose are small and an error of 1 cm. in

determining the position would not lead to as much as 5 per cent error in the tissue dose.

In connection with cross-fire, it may be well to say a little more about exit doses. If the position of the patient is such that the exit field is in contact with the treatment table, or with a firm mattress, then the exit dose is essentially the same as the tabulated depth dose for the thickness of the patient. For instance, in the example just considered, if the patient (17 cm. in anterior-posterior measurement) is lying flat on the table, the exit dose for either field is essentially the depth dose at 17 cm.—15 per cent in this case. If, however, the patient is in such a position that only air is in contact with the exit field, the exit dose is less than the tabulated depth dose, because of the lack of scattering material beyond the exit field. For this situation, certain rules have been formulated, based on experiment:

1. For small fields (10 sq. cm.) the exit dose for any thickness of part is about 90 per cent of the regular depth dose at the same level.

2. For intermediate fields (75 sq. cm.) the exit dose is from 70 to 80 per cent of the regular depth dose, depending on the thickness of the part. No appreciable error is introduced into dosage calculations by taking an average and using 75 per cent for all thicknesses, for fields of this order of magnitude.

3. For large fields (250 sq. cm.) the exit dose is from 65 to 75 per cent of the regular depth dose. Here also it is satisfactory to use the average value of 70 per cent for all thicknesses.

In comparing the relative advantages and disadvantages of various qualities of radiation, the effects of both the back-scatter and exit dose must be kept in mind. It is sometimes taken for granted that increasing the hardness of the beam must increase the absolute depth dose. This, of course, may not be true if the back-scatter for the harder beam is considerably less than for the softer. In this case, a shift to a harder radiation, with the same number of roentgens *measured in air*, may actually result in a decreased tissue dose. That this fact is frequently overlooked is demonstrated by the number of radiologists who state that their patients tolerate irradiation better with 200 kv. and 2.0 mm. copper filter than with 200 kv. and 0.5 mm. copper, and on being questioned, admit that they are specifying all doses *in air*. While it is probable that there is a certain quality effect of this sort, it must be pointed out that for the 2.0 mm. copper filter and large fields, the skin dose for a given air dose is about 10 per cent less than with 0.5 mm. copper. Hence, in addition to any possible quality difference, the skin is actually receiving less radiation, and is also receiving it at a slower rate (due to the diminished output with heavier filter). The curves of Figure 2 bring out these statements.

In Figure 7 are developed comparisons of the three hardest beams under discussion in this paper, in irradiating by cross-fire necks and pelves of certain specified dimensions. Each curve has been constructed in the same manner as the upper one in Figure 6, distances being indicated in centimeters from the midline. The upper two sets of curves in each figure show the distribution of radiation through the part when 100 r, measured on the skin with back-scatter, are delivered to each skin field; the lower sets give the doses when each side receives 100 r measured in air.

In the case of the 10-cm. neck, when incident skin doses of 100 r are employed, the total skin dose (including exit dose) for the most penetrating beam is 5 per cent more than for the next, and the dose in the midline 11 per cent higher. If the exposures for all three qualities of radiation were adjusted so as to deliver identical total skin doses, the widest variation in dose at any point within the neck, for the three qualities, would not be more than 10 per cent. Here, apparently, the radiation of choice would be the most readily available. For the larger neck, on adjusting total skin doses to equality, the hardest radiation would be found to give 10 per

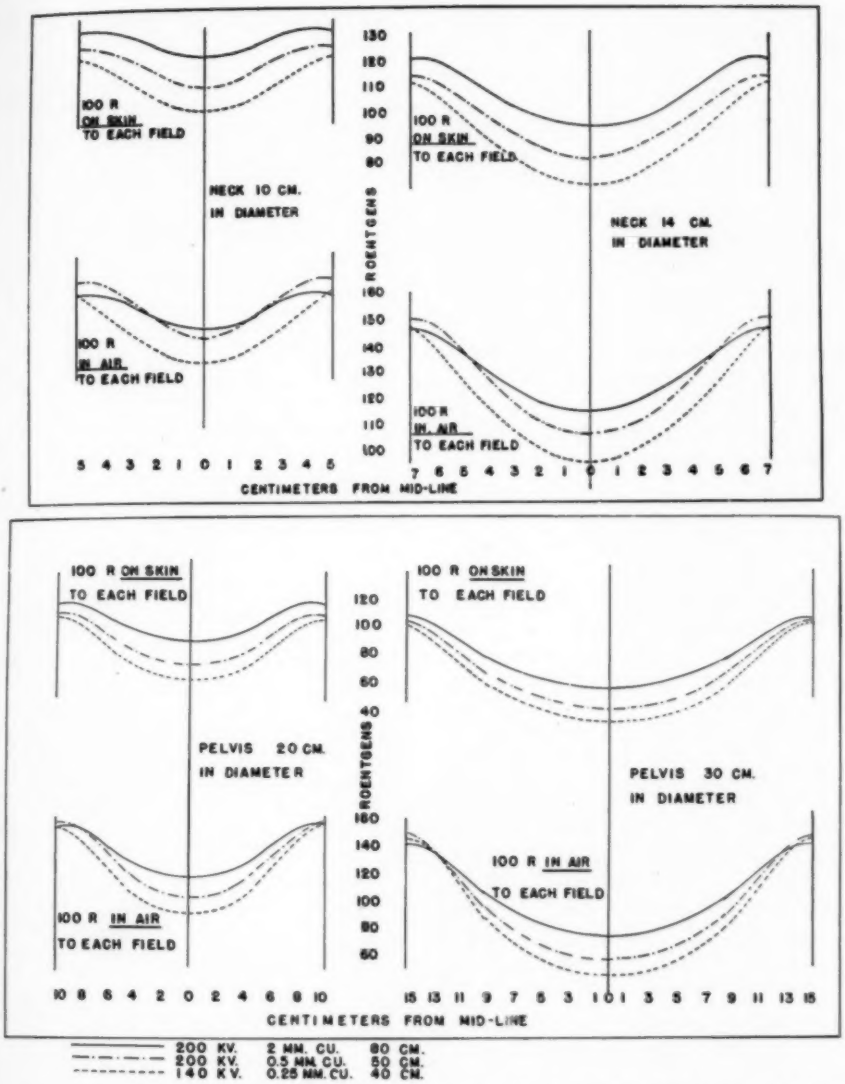


Fig. 7. Effect of variation in physical factors on distribution of radiation throughout neck, when irradiated through right and left lateral fields (100 sq. cm.), and effect of variation in physical factors on distribution of radiation throughout pelvis when irradiated through single anterior and posterior fields (200 sq. cm.)

cent more at the midline than the second quality, and 23 per cent more than the third; here the choice must lie definitely with one of the 200-kv. beams. Similar considerations applied to the chart for the pelvis indicate that this 140-kv. radiation is not satisfactory in any case. For the smaller woman, the use of the heavier copper filter and the longer distance increases the midline dose by 20 per cent, while for the larger individual the increase is almost 40 per cent. Granted that it will require much longer to deliver the dose in the second case, it may still be desirable.

Such discussions as the above bring out the fact that, if the only therapeutic equip-

ment available is a low-voltage machine, treatments should be confined to superficial lesions. An attempt to treat a deep-seated lesion with low voltage, on the assumption that a little radiation is better than none, may lead to such damage to overlying structures as to prohibit any further treatment if better equipment becomes available, without in any way inhibiting the growth of the malignant lesion. Practical examples of this state of affairs can be found in any large x-ray department, where these poorly treated patients have been referred when the superficial therapy has failed.

There is also the converse problem of the radiologist who has no low-voltage apparatus available, and must do his superficial therapy with a 200-kv. installation operated at a lower voltage and shorter distance, and without external filter. Such an equipment can usually be operated at about 140 kv., and at a distance of 35 cm. When no external filter is used, the thick glass of the deep therapy tube still acts as a considerable filter at this voltage; the emitted beam has an intensity of about 150 r per minute (air) and a half-value layer of about 5 mm. aluminum. This is only slightly harder than the radiation here considered as number 3—120 kv. with 3 mm. aluminum filter. The use of 35 cm. instead of the 30 used in the curves for this quality will increase the depth dose only slightly, for the depths which would be treated by this beam. It is evident, therefore, that this radiation can reasonably be employed for all but the most superficial therapy.

SUMMARY

A group of six sets of voltage-filter-distance factors for x-ray beams has been selected, covering the region between 100-kv. unfiltered and 200-kv. highly filtered radiation. Depth dose data for these beams have been presented in such a manner as to facilitate determinations as to their relative values in any specific condition. The usefulness of the data is illustrated by the working out of various practical problems.

The author wishes to acknowledge her indebtedness to Mr. L. D. Marinelli for his co-operation in securing hitherto unpublished data on low-voltage depth doses, and to Dr. Manuel Garcia for his assistance in the experiments leading to the rules for determining exit doses.

Memorial Hospital, New York, N. Y.

REFERENCE

1. Radiological Society of North America Standardization Committee: Technical Bulletin No. 1. Radiology **35:** 138–159, 1940.

DISCUSSION

Edwin C. Ernst, M.D. (St. Louis, Mo.): I am indeed honored to discuss this paper. Without the past contributions of Edith Quimby, Failla, Marinelli, and other physicists of the Standardization Committee—Doctors Glasser, Taylor, and many others—the progress made thus far in the field of physical x-ray measurements would have been greatly curtailed. Year after year important radiation measurement studies have been presented by our esteemed essayist and colleague—Edith Quimby. This afternoon we have been privileged to listen to further valuable contributions. Few if any previous presentations could have been of greater usefulness than this simple exposition of practical x-ray dosage charts and measurements.

I can discuss only a few of the many high points with respect to the analysis of the differential air, skin, depth, and exit dose values. I know that you will all agree with me that, rather than proving helpful, complicated formulas or graphs usually disturb the average radiologist, with the result that their employment is very limited. The romance of dosimetry, as presented this afternoon, has been extremely interesting. The acceptance and application of the roentgen unit in this country have been rapid. I well remember during the early days of the American Board of Radiology examinations the relatively limited dosage knowledge possessed by the average applicant. Whenever he did finally master the definition of the roentgen unit and with reasonable accuracy could compare his milliampere minute technic with that of the roentgen unit, the radiotherapist of that day considered himself well grounded in the physics of radiology.

During the succeeding years it was gratifying to note the rapidity with which the direct roentgen unit method of x-ray measurement was employed by the average radiologist. It was only natural, therefore, that, during this period of development of these newer methods, many of us reached the pinnacle of over-standardization, thinking only in terms of multiples of thousands of roentgen air dose units, forgetting the many other im-

portant clinical and practical considerations, and inadvertently paying too much attention to skin changes and too little to the amount of radiation ultimately reaching the tumor.

Many scientific contributions and published treatises—and, unfortunately, essayists at this very meeting—have reported results in terms of 12,000 and 16,000 roentgens, supposedly representing the actual dose administered to a specific deep-seated lesion, when in fact these misleading figures were obtained by totalling the air dose to four or six ports of entry. The examples presented by Dr. Quimby uncover the fallacy of administering like dosages to patients of different sizes and weights, under the mistaken presumption that a slight increase in the superficial dose is sufficient for effective irradiation of a larger patient. Many of our roentgen institutions persist in the unfortunate routine of overstandardizing different methods of x-ray therapy, using the designations "preoperative," postoperative," or "pelvic" roentgen cycle. To me this classification seems most unfortunate, since the necessary variability of patients in size and shape should be given greater individual consideration.

It is not surprising, therefore, that surgeons and other specialists frequently advise the technician to administer the pelvic or postoperative cycle, or even suggest that the patient enter the hospital for a "few shots of x-ray." In a measure, it has been our own fault that we have permitted these conditions to exist, rather than acting in the capacity of true consultants. For many years at the Barnard Free Skin and Cancer and DePaul Hospitals, we have carefully measured and examined each patient and then prescribed the number of tissue roentgens which should reach the tumor area. If 3,500 r is the selected tumor dose, the number of ports of entry and the physical factors are increased or decreased to meet the requirements. Medical assistants and technicians co-operate in this planning and therefore have developed an added interest in properly placing the patients for therapy. In the treatment of extremely large patients, even under the most careful planning, the tumor area is short of receiving the full tumor depth dose by a thousand or more tissue roentgens. Additional ports of entry must be devised, or the penetration of the beam be increased, to irradiate large patients adequately. In such a problem these depth charts are most helpful.

Since the interest of the patient is at stake, we should become more tumor-minded, rather than "quibbling" about differences of a few hundred roentgens in the dose measured in air and directed through the individual ports of entry. The essayist has been very timely in her emphasis on the fallacy of increasing the actual tumor dose 10 or 15 per cent in large patients, when in reality it will vary in different individuals by as much as 100 per cent under a like quality of radiation. Often we are too busy to study the individual needs of our patients, much less make a careful physical examination. The charts presented refer to but six technics and this number may not be applicable to all of our requirements, but in my opinion they are sufficiently inclusive. Contact and supervoltage therapy graphs are not included.

It should also be remembered that with certain malignant tumors atypical reactions and unusual behavior are to be anticipated. Tumors may show different responses to similar roentgen dosages. In the application of any form of radiation, whether in research or practical therapy of deep-seated malignant growths, we must continue to keep detailed records of the physical factors employed, but it is equally desirable to estimate or measure directly the amount of radiation reaching the tumor area within a known period of time. In research problems, if the histologic effects of cell changes were being studied, any analysis of the results would be valueless if the report merely stated the total roentgens delivered in air and failed to include the number of roentgens reaching the pathological cells within a known period of time. From the standpoint of research, as well as practical application, every one interested in radiation therapy should make a serious effort to become acquainted with these time-saving graphs and depth dose charts.

Before closing, I wish to urge every radiologist to work with his physicist whenever a periodic calibration is being made. It is a mistake merely to accept the physicist's analysis. How can he be of service to you unless you present to him your problems and methods of roentgen application. You may be very busy at the time of his visit, but it will pay you to make the necessary arrangements in advance for spending all of your time with him during the calibration of your roentgen apparatus.

In view of the ever-increasing problems of roentgen dosimetry, if it has been possible by means of this contribution to simplify a few of the technical procedures and methods, another epoch-making advance has been achieved by Edith Quimby and her co-workers in the interest of more effective roentgen therapeutics.

The Action of Roentgen Rays on Inflammatory Conditions[1]

ARTHUR U. DESJARDINS, M.D.

Section on Therapeutic Radiology, Mayo Clinic, Rochester, Minn.

DURING THE PAST fifteen years, an increasing number of radiologists and other physicians have become interested in roentgen therapy for inflammatory conditions. Some chronic inflammations, notably of the skin and lymph nodes, had been treated with roentgen rays as early as 1896 and 1897, but this had been done empirically. Early trials of roentgen therapy for chronic inflammations were made in this country, but it is only true to say that some of these attempts were not systematically followed up and often they were unknown to all but a few. As for the treatment of acute inflammations, much of this was first carried out in various European countries, and American radiologists have been a little slow in following the lead. It was only after the World War that the treatment of chronic and especially of acute conditions became an established procedure. This was due to the work of many physicians, but special credit is due Heidenhain and Fried,[2] who treated in this manner a large number of different kinds of inflammation. Their reports have had a greater influence, perhaps, in stimulating the use of roentgen rays in the treatment of inflammations than those of any other medical writers.

There is no longer any doubt that roentgen rays often have a beneficial effect on many varieties of acute and chronic inflammation. The evidence is now so abundant that it cannot be disregarded.

It has been difficult for internists and surgeons, and for some radiologists also, to understand how an agent which has been used so largely to treat malignant tumors can also be an effective means of treating acute or chronic inflammatory processes. In malignant neoplasms, the rays act mainly by destroying malignant cells. How can this be reconciled with the treatment of inflammatory lesions? One reason for this difficulty is that so few physicians have been familiar with the experimental background of roentgen therapy and with the sensitiveness of different kinds of tissue and cells to the rays. A second reason is the great variety of inflammations for which the therapeutic value of roentgen rays has been claimed, and the lack of a convincing explanation of the action of the rays on these many different conditions. Still another reason for the hesitation of internists and surgeons to take advantage of this method of treatment for the benefit of their patients has been the fear of deleterious changes such as are not infrequently observed following the treatment of malignant tumors.

In 1931 I attempted to explain the action of the rays on acute and chronic inflammations. This explanation was based on a knowledge of the results of many experiments on animals and of the large number of clinical observations to be found in the medical publications of different countries over many years. Although somewhat crudely presented, this explanation has been widely accepted. Later, I discovered that the main points of this explanation were not original but had been advanced previously by Pordes (12, 13) and others. Since then, this explanation has been questioned, and some radiologists now think that other factors may be more important than those to which I had drawn attention. This may or may not be true. At any rate, a reconsideration of the subject may now be in order, and I am glad to have this opportunity to correct certain misleading impressions for which some of my earlier articles may have been responsible.

In considering this problem, the first

[1] Presented before the Radiological Society of North America, at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

[2] Heidenhain was a surgeon and Fried was a roentgenologist who co-operated with him and who supervised treatment of the patients.

logical step is to review the known phases of the inflammatory process. Although this process has been investigated by a large number of men over many years, numerous phases of it remain more or less obscure. All that is really known about inflammation are some of the major, outward phases which can readily be observed.

ACUTE INFLAMMATION

In an acute inflammation the earliest perceptible changes relate to the blood vessels. The first evidence of inflammatory reaction to an irritant is an early, but temporary, contraction of the blood vessels in the affected region. This is of short duration and is soon followed by expansion of the vessels, and this is accompanied by an increased flow of blood. For a limited time, the blood flows with increased rapidity through the arterioles and capillaries but after a time (half an hour to an hour) the rate of flow gradually diminishes. When this stage has been reached, capillaries which previously have not been visible, or have been hardly perceptible, can be made out clearly; they are distended and form an extensive and close network. As the rate of the blood flow diminishes, other changes affecting the leukocytes can be observed. An increasing number of leukocytes come in contact with the walls of the vessels and gradually work their way through the walls and migrate into the intercellular spaces of the affected region. At the same time, an increasing quantity of plasma from the blood seeps through the walls of the vessels and finds its way into the surrounding tissues where, as it accumulates, it separates layers of tissue and even individual cells. As the hyperemia, the seepage of plasma, and the migration of leukocytes increase, the affected region becomes engorged, some degree of edema occurs, the part becomes abnormally hot, and pain and throbbing result from the congestion caused by the increased quantity of fluid in the tissues and the infiltration of the tissues by migrating cells.

In acute inflammation, the cells which first migrate in greatest number from the blood stream into the intercellular spaces of the inflamed tissues are the polymorphonuclear leukocytes. In some forms of inflammation, notably in acute pyogenic infections, these cells may appear for a short time to be the only kind of leukocyte present; but if the inflammation continues beyond seventy-two hours, and the longer it continues after that time, the proportion of polymorphonuclear cells among the infiltrating leukocytes tends to diminish, and the proportion of other leukocytes, especially large mononuclears (the macrophages of Metchnikoff) and small monocytes (lymphocytes), tends gradually to increase. Thus in many acute inflammations the macrophages increase until they outnumber the polymorphonuclear cells. When the inflammatory process continues beyond this point, there is a gradual increase in the lymphocytes infiltrating the affected region, and the number of these cells may become considerable. Not all the leukocytes which infiltrate inflamed tissues have emigrated from the blood stream; some of them existed in the tissues as wandering cells before the inflammatory process began.

In the early stages of an acute inflammation, the polymorphonuclear leukocytes are the most active phagocytes; they destroy large numbers of bacteria or dispose, in a similar manner, of other noxious substances which may be responsible for the inflammation; but in the attempt to perform this function, many of the polymorphonuclear cells are themselves destroyed and become the so-called pus cells. As they disintegrate, these cells liberate bactericidal substances, proteolytic ferments, and thrombin, which combines with the fibrinogen of the plasma to form fibrin. When polymorphonuclear leukocytes engulf bacteria or other foreign substances, the ferments and bactericidal substances which they contain enable them to dispose of these noxious agents by intracellular digestion.

Other leukocytes which have an important phagocytic function are the large

mononuclear leukocytes. It would appear that the main function of these cells is to dispose by phagocytosis of dead bacteria, dead leukocytes, and dead tissue cells; they are not numerous during the early stages of an inflammation, but they become increasingly numerous during later stages, and their main function is to act as scavengers. As for the lymphocytes, their principal function in inflammation does not appear to be phagocytic but rather antitoxic.

An important factor in preventing an infectious agent or other cause of inflammation from extending beyond certain limits is the coagulation of the plasma and the sealing of some of the arterioles, capillaries, and lymphatics by thrombosis. Thus, bacteria and other foreign substances are fixed at the site of inflammation and cannot be carried to the regional lymph nodes as readily as in normal tissue. Menkin's (4–9) and Menkin's and Warner's experiments have shown this clearly. They have also shown that, when the bacteria were staphylococci, they became fixed as early as one hour after inoculation. When the infection was due to pneumococcus, type I, the bacteria were not fixed until six or more hours after inoculation. When the bacteria were hemolytic streptococci, they were carried to the tributary lymphatics for almost two days after onset of the inflammatory reaction.

Of equal, if not greater, importance in localizing the inflammation, in fixing bacteria and other irritants, and in preventing them from being carried to the tributary lymph nodes, is the deposition of fibrin. This is an early factor in the defensive phase of inflammation and it also plays a major part in initiating the process of repair.

One point which has puzzled pathologists for many years has been the change in the character of the leukocytes infiltrating an inflamed region after the inflammation has continued beyond a certain time. It has long been known that, after an inflammation has proceeded for two or more hours, the proportion of polymorphonuclear cells tends to diminish, and the proportion of mononuclear phagocytes gradually increases. Menkin appears to have established the fact that this change in relative proportion of infiltrating leukocytes is related to the hydrogen-ion concentration of the exudate. As the inflammation develops, the carbon dioxide capacity of the exudate diminishes and the hydrogen-ion concentration increases, and this is accompanied by a "shift in cellular composition from polymorphonuclear cells to mononuclear phagocytes." When the *p*H falls below 6.7 or 6.5, most leukocytes appear to be injured and frank suppuration ensues.

As for the liquid part of the exudate, which is made up of plasma from the blood and lymph from the tissues, this contains the antibodies, agglutinins, opsonins, and other protective substances which have been elaborated in the blood stream, as well as thrombin and fibrinogen, which play such an essential part in the formation of fibrin. The infiltrating leukocytes as well as the wandering cells, on the other hand, contain proteolytic ferments and other bacteriolytic substances.

CHRONIC INFLAMMATION

In considering chronic inflammation, it must be remembered that between an acute and a chronic inflammation there is no sharp line of demarcation, and that the distinction between the two is largely an arbitrary one adopted by writers for the sake of convenience in description. Some inflammations, such as those of tuberculosis, actinomycosis, and syphilis, are essentially chronic from the start, but other inflammations begin as an acute process and, for one reason or another, continue until they become chronic. Outstanding features of chronic inflammations are the difference in the character of the infiltrating leukocytes, the diminished tendency for fibrin to be deposited, and the presence of varying amounts of connective tissue and of products of cellular degeneration. Polymorphonuclear cells are much less numerous, lymphocytes tend to preponderate.

and a certain proportion of eosinophils and giant cells may be present. These cellular features vary greatly, however, in different stages of the same kind of inflammation as well as in different kinds of inflammation. Thus, in an acute inflammation which lasts longer than three or four days, a certain proportion of lymphocytes is likely to be found, and this proportion tends to increase gradually as the inflammatory process continues beyond this period. Another point to bear in mind is that in some forms of chronic inflammation, notably in tuberculosis and actinomycosis, individual lesions in different stages of evolution or involution may frequently be found in the same territory, sometimes side by side.

ACTION OF ROENTGEN RAYS

Any attempt to explain the action of roentgen rays on inflammations must furnish a logical relationship between this action and the different phases of the inflammatory process. The first point to consider is whether or not roentgen rays act directly on bacteria. Many experiments have shown that some varieties of bacteria, when they are exposed to sufficiently large doses of rays, are destroyed and no longer have the power to grow, but, in order to produce this effect, doses of rays much larger than those used in treating human beings usually have to be employed. And these doses are relatively much greater still when they are compared with the small doses required to treat acute inflammation, or even when compared with the somewhat larger doses used to treat chronic inflammation. Other varieties of bacteria do not appear to be influenced directly even to this extent. The action of the rays on acute inflammations cannot, therefore, be attributed to a direct bactericidal effect of the rays.

The objection has been made that thin layers of bacterial growth in a Petri dish hardly represent a test of the power of roentgen rays to influence the growth of bacteria because, it has been claimed, organisms spread in such thin layers do not allow for much scattering of the rays. While this objection may have some validity, it does not account satisfactorily for the lack of any convincing evidence that, when inflammations are exposed to a small dose of rays, the beneficial effect is due to the direct action of the rays on the bacteria. If the rays acted in this manner, evidence of direct bactericidal effect would certainly be much more abundant.

The second point is whether the rays act mainly on the body fluids or on the cells. There is evidence that the rays do act on the body fluids (blood, plasma, and lymph), but much of this evidence indicates that the action is largely indirect; in other words, most of the changes in the fluid part of the blood, in the plasma or lymph, appear to be secondary to the direct action of the rays on the blood corpuscles or on tissue cells of different kinds. It must not be inferred that no direct changes whatever are induced in the body fluids by irradiation. On the contrary, some changes almost certainly are produced, but these are less prominent and less readily observable than are the cellular changes. It is perhaps for this reason that the evidence of these effects on the body fluids is so much less abundant.

When the cellular changes caused by exposure to the rays are considered, the contrast is striking. As far as our knowledge goes, changes of this kind constitute the bulk of the evidence now available. And from what is known of these changes, there is every reason to believe that they are much more important than the largely indirect changes in the tissue fluids.

It is well established that each variety of cell in the body has a specific range of sensitiveness to roentgen rays. So true is this that it is possible to arrange the different kinds of cells according to their range of sensitiveness, and it is also possible to recognize some varieties of tumors by the sensitiveness of the cells of which they are chiefly composed. Elsewhere I have submitted part of the evidence which substantiates this contention. From this

evidence it is clear that, among the different kinds of cells in the body, the leukocytes are the most sensitive to roentgen rays. Of the leukocytes, the lymphocytes are the most sensitive, but the large mononuclears, the polymorphonuclears, and the eosinophils are only slightly less sensitive. Physicians in general, and, indeed, many radiologists, do not realize how very sensitive these cells are. Recently, for example, Pendergrass and Hodes questioned "whether the more resistant polymorphonuclear cells are destroyed by doses of 75 r to 200 r commonly used for localized infections." The direct evidence bearing on this point is relatively meager, but the indirect evidence is abundant. One has only to remember what happens to the leukocytes, including the polymorphonuclear cells, in leukemia to realize how sensitive these cells are. It is true, as pointed out above, that the lymphocytes are slightly more sensitive than the polymorphonuclears, but the difference between them in this respect is much less than is often assumed. Soon after exposure to a small or moderate dose of roentgen rays, destruction of lymphocytes in lymphoid structures and in the circulating blood can be observed. As for the polymorphonuclear cells, exposure to the same dose is often followed for a few hours by an increase in the number of these cells; this transient increase may continue from six to twenty-four hours, after which the number of the cells falls below normal. In other cases, the transient increase in the number of polymorphonuclear cells after irradiation is not observed; the cells diminish in number from the start. In either event, how great the reduction in number may be and how long it may last depend mainly on the dose of rays and on the volume of body tissue irradiated, though individual variation also is a factor of considerable importance.

In some of my early papers on the treatment of acute inflammations I placed too much emphasis on the susceptibility of the lymphocytes and on destruction of these cells as perhaps the major factor in the effect of the rays on lesions of this kind. Since then, I have attempted to correct this misleading impression. During the early phase of acute inflammations the polymorphonuclear cells preponderate. As the inflammatory process continues, the mononuclear macrophages gradually increase in number, and later still, when the inflammation extends beyond a few days, an increasing number of lymphocytes may also be present. The more acute the inflammation and the shorter its course, therefore, the greater the likelihood of a preponderance of polymorphonuclear cells and mononuclear macrophages; and the more prolonged and less acute the inflammation, the greater the proportion of lymphocytes.

Anyone who undertakes to analyze the experimental evidence soon becomes aware that roentgen rays not only have a decidedly deleterious action on these important varieties of leukocytes, but that this action is surprisingly rapid. When the effect of irradiation on inflammatory lesions is closely observed, the rate and manner of their response are often so similar to that of the leukocytes under experimental conditions that a relationship between the two is almost inescapable.

What happens to the leukocytes which infiltrate an inflamed region when this region is exposed to roentgen rays? Certainly, there is no good reason to think that leukocytes would or could behave differently under these circumstances than they are known to behave in the body of an animal or human being under experimental conditions. It is probable that a proportion of these leukocytes are destroyed, and the cells disintegrate just as effectively as the same varieties of cells have been shown to disintegrate in the body of an animal following experimental irradiation. And the contents of the destroyed leukocytes, including the bacteriolytic substances which they contain, are scattered between the remaining intact cells and in the tissue fluids of the inflamed region.

As the disintegration of leukocytes pro-

ceeds, this is accompanied or followed by an increase in phagocytosis, which appears to be an indirect effect. As observed in different experiments on normal tissues, some of the reticular cells assume a phagocytic property. Whether or not a similar effect occurs in an inflammatory process is not clear, but at least there is no evidence that such an effect does not occur. However, the notable increase in phagocytosis by polymorphonuclear cells and mononuclear macrophages, independent of irradiation, would make it difficult to recognize any increase in this factor which might be secondary to the cellular action of the rays.

It has long been known that in inflammations caused by streptococci, notably by hemolytic streptococci, leukocytic infiltration is much less pronounced than when the inflammation is caused by staphylococci. Not infrequently, in fact, leukocytic infiltration is hardly perceptible. Much the same is true in other inflammations caused by virulent microorganisms, such as *Clostridium welchii*. An interesting point is that these inflammations do not respond so well when exposed to a single dose of roentgen rays but appear to do much better when exposed repeatedly to small doses at short intervals. Proof that this difference in the effect of irradiation is related to the relative paucity of infiltrating leukocytes has not yet been presented, but the circumstantial evidence suggests that such a relationship may well exist.

Recently, Pendergrass and Hodes have expressed the opinion that the action of roentgen rays on acute infections is due to vascular changes induced by irradiation rather than to a leukocytolytic action. They admit that "in the chronic inflammatory processes, however, in which larger total doses of radiation are usually employed, the destruction of leucocytes and young fibroblasts may be of significance." This would imply a belief that relatively large doses of radiation are required to destroy leukocytes, and that the inability to accept the idea that the main action of the rays in acute inflammations is basically the same is partly because acute inflammations are rapidly influenced by doses which, to them, seem too small to destroy leukocytes.

The conception that the action of roentgen rays on acute and on chronic inflammations rests on a different fundamental basis seems irreconcilable with the known facts. It is true that chronic inflammations respond best to doses somewhat larger than those required for acute inflammations. In the former, moreover, the treatment must be repeated a number of times at suitable intervals, while in the latter a single exposure to a small dose is often sufficient. As is well known, chronic inflammations are characterized by varying degrees of leukocytic infiltration, proliferation of connective tissue, and hyaline, caseous, or calcareous degeneration. In all probability, the relative proportion of these factors has an important bearing on the action of the rays. Since it is the product of cellular degeneration, caseous or calcareous degeneration should not be influenced by irradiation, and this is precisely what is observed in practice. Connective-tissue cells are much less sensitive to the rays than polymorphonuclear leukocytes, large mononuclears, or lymphocytes. Correlation of these several factors seems to indicate that the greater the degree of leukocytic infiltration, the more marked and the more rapid is the influence of treatment, and *vice versa*. It seems probable that leukocytic infiltration on the one hand, and connective tissue proliferation on the other, act in opposite directions, the former tending to increase the effect of irradiation and the latter tending to diminish or retard this effect.

I have already indicated that the leukocytes are much more sensitive to irradiation than most physicians realize. To make this clear would require a detailed analysis of many experiments and of many clinical observations. This, however, is impossible in a paper of this kind. Moreover, much of this evidence has been submitted elsewhere. But anyone who will

take the trouble to read carefully Warthin's report of his experiments cannot fail to be impressed. By arranging his histologic technic so that sections for microscopic study could be prepared within fifteen minutes after exposure to the rays, Warthin was able to observe disintegration of leukocytes as early as this. To become perceptible within such a short time the disintegration of the cells must have started as soon as the exposure began and must have proceeded rapidly. To speak of a latent period under these circumstances is merely to deceive oneself. In the case of leukemia a moderate dose of rays may cause the number of leukocytes in the circulating blood to diminish 50,000 or even 100,000 per cubic millimeter of blood in a single day. In spite of facts such as these, and many others, some physicians cannot recognize the great sensitiveness of leukocytes to roentgen rays.

Pendergrass and Hodes referred to experiments carried out by Hodes and Griffith but, since these experiments have not yet been published, it is impossible to analyze them and to determine their possible significance. It is possible that vascular changes and the resulting increase in blood flow through the capillary plexus of the inflamed region may have an important, and perhaps even a fundamental, bearing on the problem of the action of roentgen rays on acute inflammatory lesions. But if irradiation of an acute inflammatory process causes vascular changes and if the beneficial effects of the rays are due to these changes, more pronounced changes of the same kind should occur after the larger doses used in the treatment of chronic inflammation, and chronic inflammations should therefore respond even more rapidly than acute inflammations. Unfortunately, as every therapeutic radiologist is well aware, this is not true.

The varieties of cells of which blood vessels are composed are, in comparison with leukocytes, much less sensitive to roentgen rays. A direct action on the endothelial lining or on the connective-tissue cells of the media of the vessels requires a dose of rays sufficient to tax the tolerance of the overlying skin. It seems extremely doubtful, therefore, that the small or moderate doses used in treating inflammations could have a direct action on the cellular components of the blood vessels. Some indirect action may be produced, but convincing evidence has not yet been furnished.

Mayo Clinic, Rochester, Minn.

REFERENCES

1. DESJARDINS, A. U.: Radiotherapy for Inflammatory Conditions. J. A. M. A. **96:** 401–408, Feb. 7, 1931.

2. HEIDENHAIN, LOTHAR: Röntgenbestrahlung und Entzündung. Strahlentherapie **24:** 37–51, 1926.

3. HEIDENHAIN, L., AND FRIED, C.: Röntgenbestrahlung und Entzündung. Arch. f. klin. Chir. **133:** 624–655, April 26, 1924; Röntgenstrahlung und Entzündung. Klin. Wchnschr. **3:** 1121–1122, June 1924.

4. MENKIN, VALY: Studies on Inflammation. I. Fixation of Vital Dyes in Inflamed Areas. J. Exper. Med. **50:** 171–180, August 1929.

5. MENKIN, VALY: Studies on Inflammation. IV. Fixation of Foreign Protein at Site of Inflammation. J. Exper. Med. **52:** 201–213, August 1930.

6. MENKIN, VALY: Studies on Inflammation. V. Mechanism of Fixation by the Inflammatory Reaction. J. Exper. Med. **53:** 171–178, February 1931.

7. MENKIN, VALY: Studies on Inflammation. VII. Fixation of Bacteria and of Particulate Matter at the Site of Inflammation. J. Exper. Med. **53:** 647–660, May 1931.

8. MENKIN, VALY: Studies on Inflammation. IX. A Factor in the Mechanism of Invasiveness by Pyogenic Bacteria. J. Exper. Med. **57:** 977–991, June 1933.

9. MENKIN, VALY: Studies on Inflammation. X. The Cytological Picture of an Inflammatory Exudate in Relation to Its Hydrogen Ion Concentration. Am. J. Path. **10:** 193–210, March 1934.

10. MENKIN, VALY, AND WARNER, CHARLOTTE R.: Studies on Inflammation. XIII. Carbohydrate Metabolism, Local Acidosis, and the Cytological Picture in Inflammation. Am. J. Path. **13:** 25–43, January 1937.

11. PENDERGRASS, E. P., AND HODES, P. J.: Roentgen Irradiation in the Treatment of Inflammations. Am. J. Roentgenol. **45:** 74–106, January 1941.

12. PORDES, F.: Ueber die Natur der Wirkung der Röntgenstrahlen, speziell über das Verschwinden von Anurie nach Nierenbestrahlung. Wien. klin. Wchnschr. **36:** 656–657, Sept. 13, 1923.

13. PORDES, F.: Der Mechanismus der Röntgenwirkung; ein Erklärungsversuch. Fortschr. a. d. Geb. d. Röntgenstrahlen **31:** 287–297, 1923–1924.

14. WARTHIN, A. S.: An Experimental Study of the Effects of Roentgen Rays upon the Blood-Forming Organs, with Special Reference to the Treatment of Leukemia. Internat. Clin., series 15. **4:** 243–277, 1905.

The Value of Roentgen Therapy in Pneumonia Which Fails to Respond to the Sulfonamides[1]

J. P. ROUSSEAU, M.D., W. M. JOHNSON, M.D., and GEORGE T. HARRELL, M.D.[2]
Winston-Salem, N. C.

THE VALUE OF irradiation in some of the acute and chronic inflammations can no longer be doubted by anyone familiar with the repeated favorable response observed when this method of treatment is judiciously employed.

While much valuable scientific and experimental research on the effects of irradiation in inflammatory tissue has been done, the exact mechanism of its action in infections has not as yet been made clear. This, however, does not in the least detract from its practical value nor should it deter us from its use in those cases in which it is indicated.

The valuable observations of Fried (1), Desjardins (2, 3), Hodges (4), Quimby and Quimby (5), and Pendergrass and Hodes (6) have contributed much to our understanding of the favorable reactions observed with roentgen therapy in the infections. Within recent years, the reports of Powell (7, 8, 9), Fried (10), Edsall and Pemberton (11), Merritt and McPeak (12), Solis-Cohen and Levine (13), and Settle (14) have aroused much interest in this method of treating pneumonia. These authors have shown favorable and uniform results in well controlled groups of lobar pneumonia patients treated with small doses of x-ray. The fundamental principles involved are the same as those for the x-ray treatment of any of the other acute infections.

The present paper will be limited to certain observations made on the analysis of two series of acute lobar pneumonia patients treated since 1937. In the first series no specific drug or serum was used in any case. Only x-rays and the usual supportive and symptomatic measures were employed. In the second series, x-ray treatment was used only after what seemed to be a thorough trial of sulfonamide therapy, over a period of not less than three days or more than seven days, plus the usual routine medical care.

In the winters of 1937, 1938, and 1939, we treated with small doses of x-ray, at the City Memorial, Reynolds Colored Memorial, and North Carolina Baptist Hospitals, 176 unselected and consecutive cases of pneumonia. Seventy-two of these patients were found by repeated sputum and roentgen examinations to have atypical bronchopneumonia or lobar pneumonia due to non-pneumococcic and mixed infections. The remaining 104 patients had acute pneumococcic lobar pneumonia. A pneumococcus of one type or another was found to be the predominating organism in the sputum, which was typed by the Neufeld method.

Inasmuch as our chief interest in the treatment of pneumonia centered around the pneumococcic type, all other cases are eliminated from this report by strict adherence to the following criteria: (*a*) a positive clinical diagnosis by the attending physician and resident medical staff; (*b*) confirmation of the diagnosis by bedside x-ray film; (*c*) additional confirmation by recovery of the pneumococcus from the sputum and typing.

The roentgen treatment was given as soon as possible after admission to the hospital. The technic was the same in general plan as that used by Powell, Solis-Cohen, Settle, and others. The factors were 120 kv.p., 3 mm. aluminum filter, 40 cm. T.S.D., 18 r (in air) per minute, 200 r (in air) at a single dose, repeated

[1] From The Bowman Gray School of Medicine, Wake Forest College, Winston-Salem, N. C. Presented before the Radiological Society of North America, at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

[2] Professor of Radiology, Clinical Professor of Medicine, and Assistant Professor of Medicine, respectively.

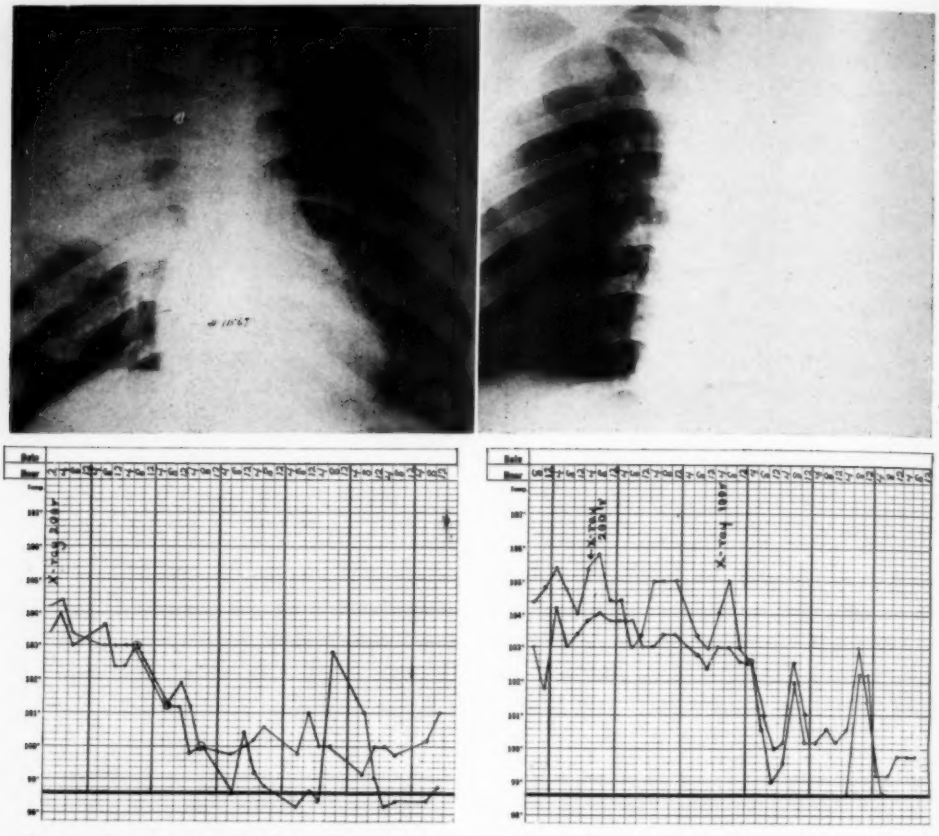


Fig. 1. Lobar pneumococcic pneumonia; 200 roentgens to anterior right chest immediately after admission. Prompt response to irradiation, with complete recovery, temperature, pulse, and respiration showing gradual decline to normal over three-day interval.

Fig. 2. Massive lobar pneumonia of left lung; patient ill three days before admission; 200 roentgens on second hospital day at 4 P.M., followed by clinical improvement but little change in temperature. Second treatment, 24 hours later, followed by rapid lysis and complete recovery.

at intervals of thirty-six hours, if necessary, for one, two, or three doses. The field was large enough to cover a wide area of uninvolved lung around the consolidated area shown on the x-ray film. An anterior port was used for the first treatment and a posterior port for the second treatment. All patients were treated in bed so that they would not be disturbed any more than was necessary.

The following post-radiation procedures were employed: daily white blood cell count and Schilling differential count; repeated blood cultures for the duration of the illness; repeated bed-side films of the chest to show progress of consolidation and resolution; daily clinical and physical examinations by the medical staff:

Statistical analysis of this group shows:

Total number of cases.....	104
Recovered.....	98
Died.....	6
Mortality rate.....	5.7%
Age.....	9 weeks to 94 years
Average age.....	29.8 years
Average duration of illness before treatment.....	2 days

From this analysis, together with certain clinical observations, the following conclusions seem justified:

1. The mortality rate in this series of cases (5.7 per cent) was considerably

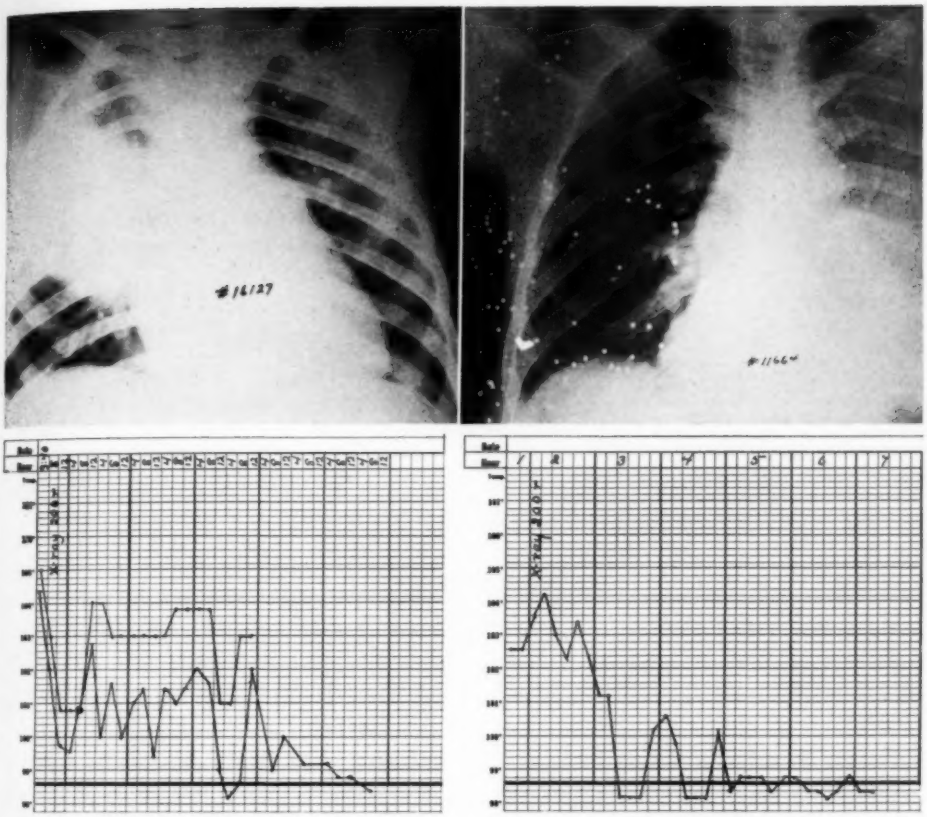


Fig. 3. Lobar pneumonia; 200 roentgens to anterior chest immediately after admission, followed by clinical improvement. Temperature normal on fourth hospital day.

Fig. 4. Left lower lobe pneumonia; 200 roentgens to left anterior chest on the second hospital day. Prompt response: rapid lysis and complete recovery.

lower than the previous ten-year mortality rate of 28 per cent for pneumonia in our community.

2. Improvement was often noted by the patient before any alteration in the temperature, pulse rate, and physical findings could be detected. Many patients stated that they felt better, coughed less, and had less pain in the chest within six to twelve hours after treatment.
3. A fall in the temperature, pulse, and respiratory rate was usually manifested in twelve to thirty-six hours after the first treatment. The earlier the treatment was started the more favorable appeared to be the response. The temperature fell by rapid lysis in the majority of cases, reaching normal within twenty-four to forty-eight hours after the initial decline.
4. The leukocyte count decreased simultaneously with the fall in the temperature.
5. Clinical improvement was well developed before any change could be demonstrated by x-ray in the consolidated area.
6. Resolution of the consolidation seemed to be similar to the resolution occurring in patients treated by any other method.
7. The presence of granulopenia was definitely a bad prognostic sign. Both of the 2 patients showing a

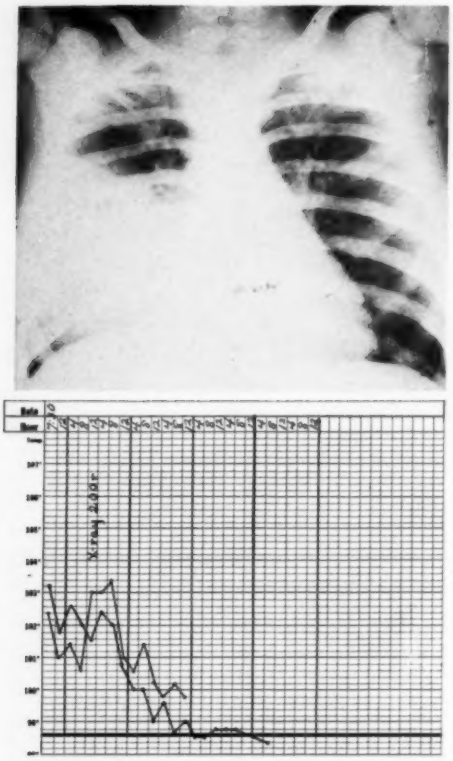


Fig. 5. Acute lobar pneumococcic pneumonia; 200 roentgens to anterior chest second hospital day. Prompt response. Temperature decline by rapid lysis. Complete recovery.

shift to the left in differential count died.

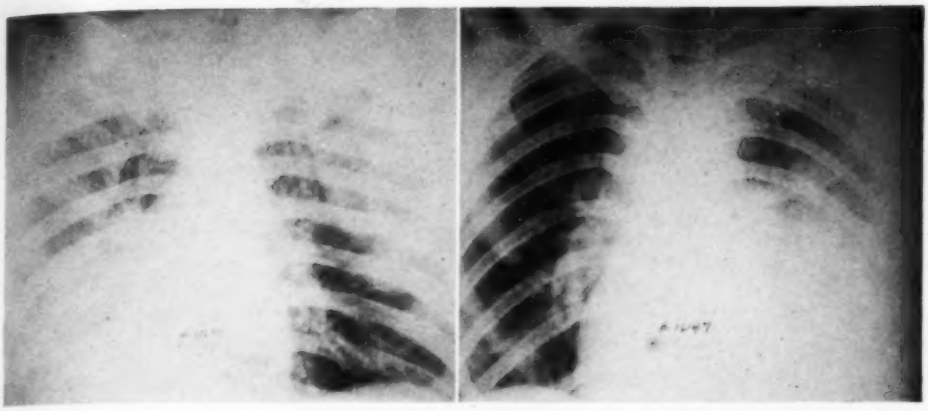
8. The presence of bacteremia was likewise a grave prognostic sign. Four of 9 patients with bacteremia died; 5 recovered, but the pneumococcus in these was of the higher types and was thought to be less virulent. All deaths in this series occurred in patients with bacteremia, granulopenia, or both.
9. There is no known artificial method of stimulating the bone marrow to produce leukocytes in the patients with granulopenia. In our granulopenic cases, sterile turpentine abscesses, pentnucleotides, liver extract, and foreign protein injections failed to increase the total white cell count.
10. The convalescence time in this series was reduced to about half that usually observed in pneumonia. The average stay in the hospital was 12.5 days.
11. No deleterious or untoward reactions from the radiation treatment could be detected locally, in the skin, lungs, or blood stream in any of the cases.

Figures 1, 2, 3, 4, and 5 are x-ray films of the chests, with accompanying reproductions of the temperature charts, in representative cases in this series, selected to illustrate the progress of the disease in patients responding favorably to roentgen treatment. Figures 6 and 7 are typical illustrations of cases in which death occurred and in which no favorable effect whatever could be detected at any time after roentgen treatment was given. Both these patients had granulopenia with a shift to the left, and one had bacteremia. Each received only one x-ray treatment, because of the fear that radiation might still further reduce the total white cell count.

Since 1939, when the sulfonamides came into general use, and proved so effective in the treatment of pneumonia, we have had but few requests for roentgen therapy. We have treated occasional cases of pneumonia in which the sulfonamides had not been used, and in these the results continue to compare favorably with those in the series analyzed above.

We have fortunately been able to collect a small series of patients treated with x-ray who had previously been energetically treated for a period of from three to seven days with a sulfonamide. In this group of cases, it was evident that the course of the disease had not been favorably affected in any way by sulfonamide therapy. The patients were growing rapidly and progressively worse with adequate doses of the drug. By all clinical standards, it appeared that death was inevitable in all cases in this group.

The methods of establishing and proving



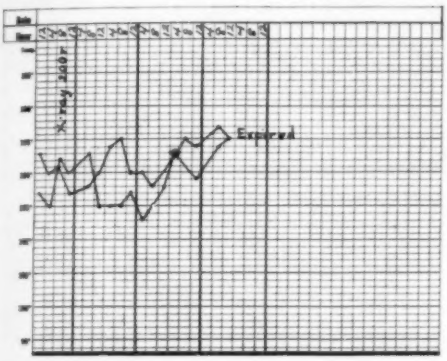


Fig. 6. Left lower lobe pneumonia; patient critically ill; 200 roentgens to left anterior chest immediately after admission. Death on fourth hospital day. At no time was there any evidence of improvement in clinical symptoms. Treatment was not repeated because of leukopenia and marked granulopenia. The temperature chart is characteristic of all fatal cases in this series.

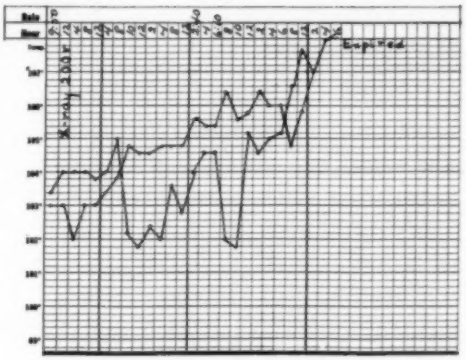


Fig. 7. Left lower lobe pneumonia; 200 roentgens to anterior chest wall immediately after admission. No improvement noted. Patient grew rapidly worse and expired on fourth hospital day. This patient had pneumococcus type III, granulopenia, and a positive blood culture. As in the case shown in Figure 6, treatment was not repeated, because of the granulopenia.

the diagnosis of pneumococcic lobar pneumonia followed in the first group were strictly followed in this second small series of cases. The same post-radiation methods of study were also followed.

Statistical data for this group are as follows:

Total number of cases	29
Recovered	22
Died	7
Mortality rate	27.1%
Ages	2 to 78 years
Average age	31 years
Duration of illness before treatment	3 to 7 days
Average duration of illness before treatment	5.8

The close observation of these patients seems to justify the following conclusions:

1. Although the effectiveness and simplicity of sulfonamide therapy entitle it to first place in the treatment of pneumonia, x-ray treatment appears in some cases to have distinct advantages. One such group of patients consists of those who are known by previous experience to tolerate the sulfonamides badly. Another group is composed of the very old, the debilitated, and those with serious heart, kidney, or liver disease, in whom the toxic effects of the drug may turn the scale against recovery. In any of these,

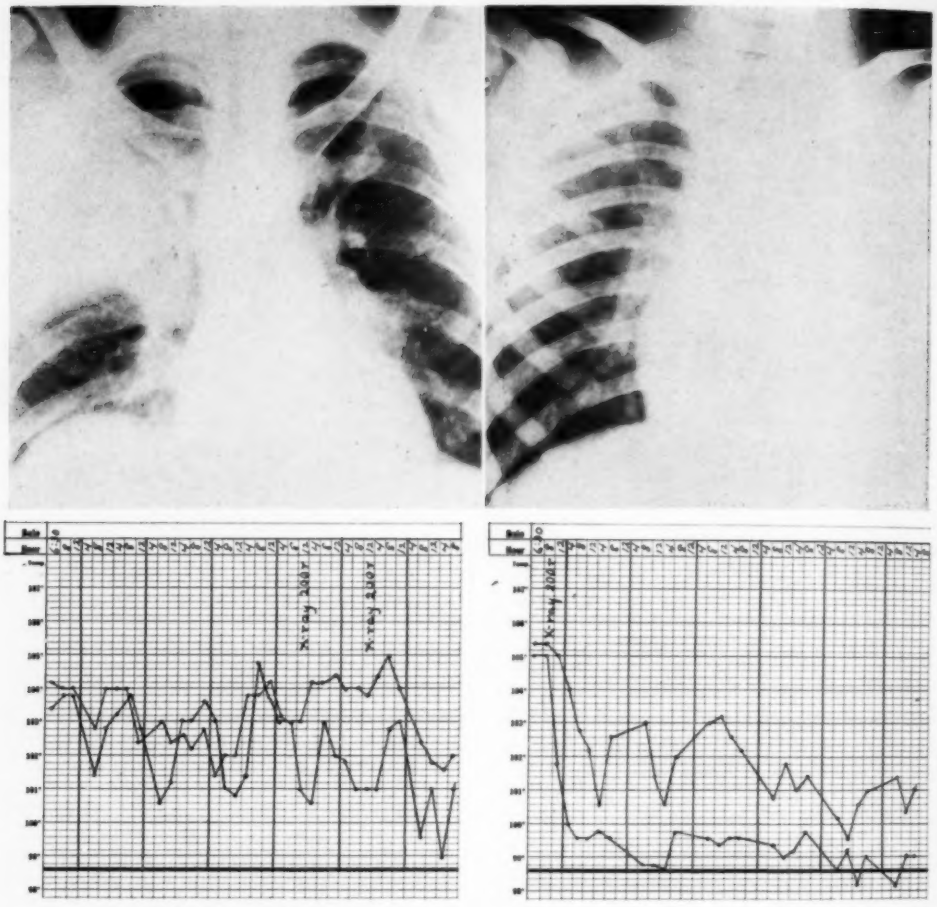


Fig. 8. Lobar pneumonia of right lung. Sulfonamide therapy for first four hospital days with no improvement. Irradiation, 200 roentgens to right anterior chest, at noon on fourth hospital day, and a second roentgen treatment 24 hours later, with usual response and full recovery by rapid lysis.

Fig. 9. Massive lobar pneumonia of left lung. Patient treated five days with sulfonamides before admission to hospital. Irradiation with 200 roentgens to left anterior chest wall immediately after admission. Prompt recovery. Fall in temperature and pulse, and clinical improvement by slow lysis.

but especially in the aged, the dehydration and inanition resulting from intense nausea or from simple anorexia induced by the sulfonamide may bring about a fatal outcome, even after defervescence. X-ray therapy has been found strikingly free from any toxic side-effects and the mortality rate with this form of treatment compares favorably with that in the groups treated by the sulfonamides or by serum.

2. The sulfonamides are of great value in the treatment of pneumococcic pneumonia, but the mortality rate still remains too high. It may be argued that some or all of the patients in this series continued to be ill because of sulfonamide fever. If this were true, they should not have shown all the clinical evidence of severe pneumonic illness together with consolidation on the x-ray film, in which no evidence of resolution could be de-

tected. No patient in this series had been treated with a sulfonamide longer than seven days, and it is our impression that sulfonamide fever rarely develops before ten to twelve days of treatment.

3. The response to roentgen treatment in this series was the same as that previously noted in cases in which the sulfonamides had not been used. The change for the better, as evidenced by a fall in the temperature, pulse, respiratory rate, and white blood count, occurred about the same number of hours after the treatment, and resolution of the consolidation in the lungs progressed in the same manner.
4. No ill effects from x-ray could be detected in patients already saturated with sulfonamide drugs for a period of three to seven days. Because of Kelly's warning that the combination of x-ray and sulfonamides in the treatment of infections might be harmful, the drug was discontinued immediately upon making the decision to try x-ray treatment.
5. Perhaps a few patients are kept on sulfonamides too long, when by the addition of x-ray some of them might be saved.
6. Analysis of the statistical data on page 285, shows that 79 per cent of 29 patients who obviously were not responding to chemotherapy recovered after the administration of x-rays.
7. The presence of bacteremia and granulopenia was again found to be of the gravest prognostic significance. All patients with bacteremia and granulopenia died.
8. In all the patients who died, the interval elapsing between the x-ray treatment and death was less than fifteen hours. Three died within one hour, one died in the fifth hour, two in the tenth hour, and one in the fifteenth hour. All patients surviving as long as fifteen hours after the treatment completely recovered. This suggests, first, that many of these cases were hopeless before institution of treatment and, secondly, that x-ray may not exert its maximum effect on the course of the disease for fifteen to twenty-four hours. This observation is, we believe, borne out by the fact that changes in the temperature, pulse, respiration, and white blood count are usually not demonstrable until fifteen to thirty-six hours after x-ray treatment.
9. There is apparently no contraindication to the use of x-rays in patients with pneumonia who have previously received therapeutic doses of a sulfonamide. We believe that the drug

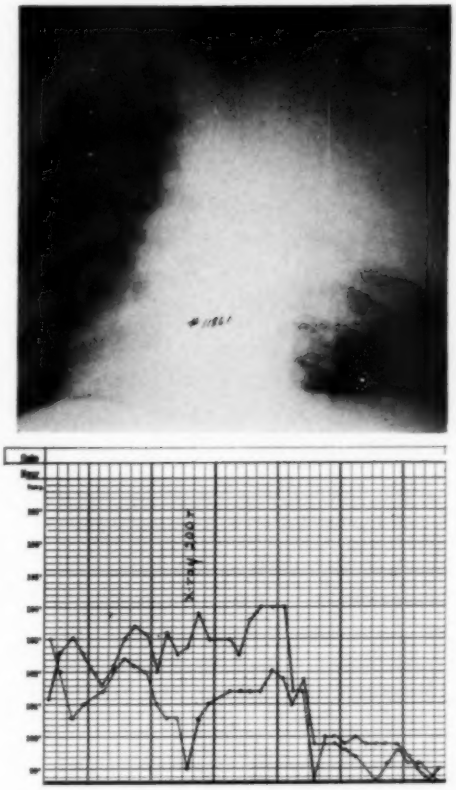


Fig. 10. Lobar pneumonia of right upper lobe, treated two days before admission and two days after admission with a sulfonamide. No response noted. On third hospital day, 200 roentgens to right anterior chest, followed within 24 hours by clinical improvement and rapid lysis. Complete recovery.

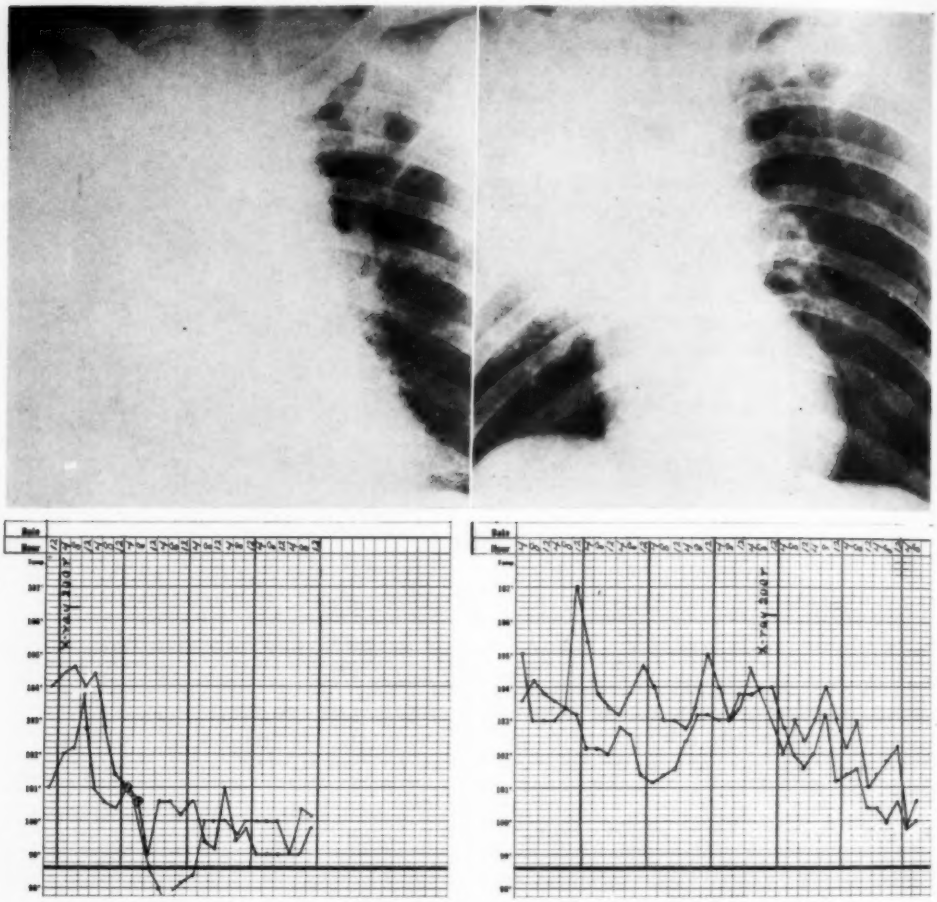


Fig. 11. Massive lobar consolidation of right lung. Treatment with sulfonamides four days before admission to hospital. No improvement. Irradiation—200 roentgens to right anterior chest—on second hospital day, followed by rapid lysis and complete recovery.

Fig. 12 Lobar consolidation of right upper lobe. Patient ill two days before admission and treated with sulfonamides for four days in the hospital without improvement. Blood culture positive. Pneumococcus type VII. Immediately following x-ray treatment there was a gradual step-ladder like decline in temperature, with complete recovery, the temperature reaching normal with a gradual daily decline over a period of three days following irradiation.

should be stopped when irradiation is instituted, until more is known about the biological, physiological, chemical, and immunological reactions of both these agents.

Figures 8, 9, 10, 11, and 12 are films and reproductions of the temperature charts in 5 typical cases in this series with recovery. By comparison with the films and temperature charts in the first series, the close similarity in the response of the two groups of cases is clearly demonstrated.

While this series is admittedly small, the assumption that roentgen therapy is of definite value in pneumonia patients who fail to show a satisfactory response to sulfonamide therapy is justifiable.

The action of x-rays in acute infections is not as yet satisfactorily explained. Many theories have been advanced by various investigators, and sooner or later conclusive proof of how and why small doses of x-ray have a favorable influence on inflammatory processes will be estab-

lished. In 1938 we started some experimental work on animals in the treatment of pneumonia, and one of us (Harrell) is continuing this research with pneumococcic infections in the laboratory animal. We hope that this will, in time, throw additional light on this complex problem.

726 Nissen Building
Winston-Salem, N. C.

BIBLIOGRAPHY

1. FRIED, C.: Die artefizielle Pneumonie und ihre Bestrahlung. Strahlentherapie **58:** 430–448, 1937.
2. DESJARDINS, A. U.: Dosage and Method of Roentgen Therapy for Inflammatory Conditions. Radiology **32:** 699–707, June 1939.
3. DESJARDINS, A. U.: Radiotherapy for Inflammatory Conditions. J. A. M. A. **96:** 401–408, Feb. 7, 1931.
4. HODGES, F. M.: Roentgen Therapy of Certain Infections. Am. J. Roentgenol. **35:** 145–155, February 1936.
5. QUIMBY, A. J., AND QUIMBY, W. A.: Unresolved Pneumonia: Successful Treatment by Roentgen Ray. New York State J. Med. **103:** 681, 1916.
6. PENDERGRASS, EUGENE P., AND HODES, PHILIP J.: Roentgen Irradiation in Treatment of Inflammations. Am. J. Roentgenol. **45:** 74–106, January 1941.
7. POWELL, E. V.: Radiation Therapy of Lobar Pneumonia. Texas State J. Med. **32:** 237–240, July 1936.
8. POWELL, E. V.: Roentgen Therapy of Lobar Pneumonia. J. A. M. A. **110:** 19–22, Jan. 1, 1938.
9. POWELL, E. V.: Treatment of Acute Pneumonia with Roentgen Rays. Am. J. Roentgenol. **41:** 404–414, March, 1939.
10. FRIED, C.: The Roentgen Treatment of Experimental Pneumonia in the Guinea-Pig. Radiology **37:** 197–204, August 1941.
11. EDSALL, D. L., AND PEMBERTON, R.: Use of X-rays in Unresolved Pneumonia. Am. J. M. Sc. **132:** 286–297, 1907.
12. MERRITT, E. A., AND MCPEAK, E. M.: Roentgen Irradiation in Unresolved Pneumonia. Am. J. Roentgenol. **23:** 45–48, January 1930.
13. SOLIS-COHEN, L., AND LEVINE, S.: Roentgen Treatment of Lobar Pneumonia. Am. J. Roentgenol. **42:** 411–417, September 1939.
14. SETTLE, EMMETT B.: Roentgen Treatment of Lobar Pneumonia. Am. J. Roentgenol. **45:** 591–599, April 1941.

Roentgen Therapy for Acute Sinusitis[1]

WALTER C. POPP, M.D.,[2] and HENRY L. WILLIAMS, M.D.[3]

Mayo Clinic, Rochester, Minn.

IN JUNE 1940 we presented a preliminary report of our experience in the use of roentgen therapy for acute sinusitis (6). We have since continued the use of this method of treatment and shall present in this paper a comparative study of the preliminary report and recent results. Since the treatment of acute sinusitis with roentgen rays is relatively new, we believe that a brief review of the literature is indicated.

Dysart (2) in 1939 and Gatewood (3) in 1940 published extensive reviews of the literature on the use of roentgen therapy in otolaryngology. Dysart, who was seemingly in favor of the method, was able to discover many reports favorable to this mode of treatment. Gatewood, on the other hand, reported many unfavorable observations. It is interesting to note that only in an article by Heidenhain and Fried did either author duplicate the other's review. For this reason one article is an excellent supplement to the other; between them they cover the literature so thoroughly as to render any duplication of their efforts superogatory.

Dysart quoted Desjardins, and Gatewood quoted Larsell and Fenton, to the effect that roentgen treatment depends on destruction of the leukocytic infiltration in the submucosa of the sinuses. Desjardins expressed the belief that this destruction makes the protective substances within the cells more readily available for defense purposes than they are in the intact cell. Larsell and Fenton attributed the effect of roentgen rays to the destruction of the lymphocytic infiltration alone, the breaking down of which causes an increase in the number of macrophages in the submucosal tissues.

Dysart concluded that a review of the literature on roentgen therapy of inflammatory conditions shows much favorable and little unfavorable evidence. He expressed the belief that in acute conditions treatment should be started as early as possible and that small doses produce better results than large doses. He reported several cases of acute sinusitis in which roentgen irradiation was beneficial.

Gatewood, on the other hand, stated that the fact that most of the literature on the subject of the treatment of sinusitis with roentgen rays had been produced by radiologists made it difficult to evaluate the end-results reported. He felt that roentgen therapy for any form of sinusitis was in the earliest experimental stage and that it was essential for the rhinologist to make the final examination in order to avoid erroneous conclusions.

In a recent paper Kornblum (4) also reviewed the literature on roentgen therapy in sinusitis. He did not present statistical data of his own, but his conclusion, based on his experience in some 400 to 500 cases, is as follows: "Anyone who uses roentgen therapy for sinusitis with an open mind, in properly selected cases, with proper roentgen technic, and under proper clinical supervision is certain to be convinced of its effectiveness and value as an adjunct to our present therapeusis of sinusitis."

From the reviews of Dysart and Gatewood it is obvious that Dysart reported results in treatment of acute conditions, whereas Gatewood dealt with chronic conditions alone. These papers are typical of the lack of unanimity of reports on the use of roentgen therapy for sinusitis. It was thought, therefore, that a roentgenologist and a rhinologist working together might resolve some of the difficulties and doubts which previous reports had raised. Accordingly the following at-

[1] Presented before the Radiological Society of North America, at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

[2] Section on Therapeutic Radiology.

[3] Section on Otolaryngology and Rhinology.

tempt has been made to evaluate the results of applying roentgen therapy to patients suffering from acute sinusitis.

The cases in each series were divided, on clinical grounds, into moderate and severe types. Cases assumed to be of moderate type were those in which the patient would tolerate a repetition of firm pressure over the affected sinus or sinuses and in which the pain did not require morphine for relief or did not seriously interfere with sleep. Cases regarded as of severe type were those in which the patient would not willingly submit to a repetition of moderately firm pressure over the involved sinuses and in which the pain and headache were severe enough to prevent sleep or to require morphine for relief. Cases in which the patients were ill enough to require hospitalization or had an associated condition such as acute hemorrhagic nephritis or orbital abscess were also included in the severe group.

Good results implied disappearance of pain within one to six hours after the roentgen treatment, disappearance of tenderness within the same interval, disappearance of congestion from the nasal mucosa, and cessation of discharge into the nasal chambers from the sinuses. The majority of the patients received additional treatment by the Dowling pack method, although in many cases relief by this method had failed to ensue before roentgen therapy was instituted. In the cases in which the patients were ill enough to require hospitalization, hot compresses were used in addition. For a few of the more severely ill patients sulfonamide drugs also were employed.

It was thought that the patients selected for study were ill enough and had sufficiently marked symptoms so that they would offer at once a severe test of the method of therapy and an opportunity for definite evaluation. The fact that more than one therapeutic method was used in many of the cases in this series makes it necessary to confine ourselves to reporting clinical impressions rather than definite percentages of improvement. The latter feat is rendered additionally difficult by the fact that acute sinusitis tends to spontaneous recovery, that is to say that in evaluating any method of therapy in this infection one is assessing the effectiveness of the defense mechanisms of the body in addition to the effect of the specific therapy used.

The preliminary report concerned 56 patients: 31 cases were classified as severe and 25 as moderately severe. Because of reports in the literature that treatment was more effective early in the course of the infection than later on, both groups were arbitrarily divided into those in which symptoms had been present for one to five days before the beginning of treatment and those in which symptoms had been present more than five days.

In the group of cases of severe sinusitis, of those in which pain and tenderness had been present from one to five days before roentgen therapy was instituted, there were 6 in which these symptoms ceased within six hours after the first treatment and one in which pain and tenderness disappeared abruptly after the second treatment. These results were classified as good. In an additional case, in which rather heavy doses had been used, symptoms were increased for three days and then abruptly disappeared with complete cessation of nasal discharge and inflammatory swelling of the nasal mucosa on the evening of the third day. In 2 other cases, pain and tenderness continued until after the third treatment, when they suddenly disappeared. These were classified as moderately good results. In 3 cases no apparent effect from the application of roentgen therapy could be noted.

There were 18 cases in which severe sinusitis had been present for more than five days. In 9 of these pain and tenderness disappeared after the first treatment, and in 2 after the second treatment. These were assumed to be good results. Pain and tenderness disappeared in one case after the third treatment. This was regarded as a moderately good result. In 6 cases no apparent effect of roentgen therapy

could be noted. In one of these cases trephination was required because of edema over the frontal area and high fever with leukocytosis suggesting beginning frontal osteomyelitis. Another patient in this group had a complicating orbital abscess and an abscess of the frontal lobe. This patient died three months later from the effect of the frontal lobe abscess. This is the only death in the series.

Twenty-five patients were classified as having moderately severe sinusitis. These were also arbitrarily divided into a group in which symptoms of pain and tenderness had been present for one to five days before the beginning of treatment and one in which they had been present for more than five days. Of the 9 cases in which symptoms had been present for one to five days, there were 8 in which pain and tenderness disappeared after the first roentgen treatment and one in which such response was delayed until after the second treatment. In no case in this group was there failure to respond to treatment.

Of the 16 cases in which pain and tenderness had been present more than five days, there were 8 in which there was complete remission of these symptoms within six hours after the first roentgen treatment. In 6 cases only a moderately good result was obtained. One patient who received an unusually heavy dose had an increase of pain and tenderness for four days with an abrupt cessation at the end of that time of all symptoms of sinusitis, including pain, tenderness, discharge, and evidence of congestion of the nasal mucosa. In 4 other cases, although pain and tenderness were relieved, discharge into the nose seemed unusually prolonged. In another case in which the patient apparently secured complete relief after the first treatment, there was a recurrence of symptoms two weeks later which required the removal of a diseased pharyngeal bursa and submucous resection of the nasal septum. In 2 cases there was no apparent effect from roentgen therapy.

The recent survey included 43 patients. Twenty-two cases were classified as severe involvement, and 21 moderately severe. As in the preliminary survey, both groups were arbitrarily divided into those in which symptoms had been present for one to five days before the beginning of treatment and those in which symptoms had been present more than five days.

In the group of severe cases in which pain and tenderness had been present from one to five days before treatment was instituted, there were 10 in which these symptoms disappeared after the first treatment and 2 in which they disappeared abruptly after the second treatment. In 2 other cases the symptoms disappeared only after the third treatment. These were considered as fair results.

There were 8 cases in which severe sinusitis had been present more than five days. In 4 of these the symptoms disappeared after the first treatment and in 3, after the second treatment. Pain and tenderness disappeared after the third treatment in one case and this was assumed to be a fair result.

Twenty-one patients were classified as having moderately severe sinusitis. These were also arbitrarily divided into a group in which symptoms of pain and tenderness had been present for one to five days and one in which symptoms had been present more than five days before the beginning of treatment. Of the cases in which symptoms had been present from one to five days, there were 8 in which these disappeared after the first treatment and 2 in which they disappeared after the second treatment. In one case no response to treatment occurred. In 10 cases pain and tenderness had been present for more than five days. In 5 of these the symptoms disappeared after the first treatment and in one after the second treatment. Three treatments were necessary in one case. This was considered a fair result. In 3 cases there was no apparent effect from roentgen treatment.

METHOD OF TREATMENT

Because of our experience in other acute inflammatory conditions, we felt that acute

sinusitis might be treated equally well with relatively small doses of roentgen therapy. Only those sinuses giving symptoms and showing objective evidence of involvement were irradiated. If a diagnosis of pansinusitis was made, all sinuses were treated.

In cases in which symptoms had been present for one to five days, doses of 50 r were used, and in those cases in which the symptoms had been present more than five days, 75 to 100 r were given. Treatments were repeated on alternate days and numbered from one to three, depending on the symptomatic response. The treatments were given with voltages generated at 130 kv. (constant potential), filtered through 6 mm. of aluminum except for children, for whom a filtration of 4 mm. of aluminum was used, at a distance of 40 cm.

In the preliminary series, treatments were given through one field to cover both frontal areas and one field each for the maxillary sinuses, thus using three areas, the eyes and eyebrows being carefully protected. In the recent series we treated all patients through two fields, dividing the face vertically into two equal parts, using the nose as the midline. Treatments were given at an angle of about 45 degrees, converging toward the nose. The eyes were not protected while the treatment was being given. We felt that this method permitted a much more thorough irradiation of all the sinuses as well as the natural ostia.

COMPARISON OF RESULTS

In Table I it will be noted that there is a fair improvement in the results obtained in the recent series. Since the same criteria for the selection of cases were used for each series and the treatments were given under the same circumstances, we believe that the improvement in the percentage of good results is the consequence of rearrangement of the fields of exposure.

TABLE I: COMPARISON OF RESULTS IN TWO SERIES OF CASES

	Total Cases	Good Results
Both series	99	70 (70.7%)
Comparison according to severity of cases		
Moderately severe	46	33 (71.7%)
Severe	53	37 (69.8%)
Comparison of former and recent experience		
Former	56	35 (62.5%)
Recent	43	35 (81.4%)

CLINICAL IMPRESSIONS

Relief of pain and headache is the most striking effect of roentgen therapy in acute sinusitis. Such relief is noted early in cases in which there are favorable results. The symptomatic improvement is usually accompanied by a noticeable increase of discharge, so that the effect of roentgen therapy on the sinuses may be due to diminishing the engorgement of the nasal mucosa. This effect is definitely observable. More good results seemed to be obtainable when therapy was instituted early, and the results seemed to be better in an initial attack of sinusitis than in a recurrence after previous attacks. Although not implicit in the material presented, there seemed to be an effect of preventing recurrences in patients who had previously been subject to repeated attacks of acute sinusitis. While roentgen therapy can by no means be relied on as the sole treatment of acute sinusitis, it has been shown to be a very useful adjunct. The recent survey indicates that thorough exposure of the natural ostia as well as the sinuses to roentgen rays is highly important in obtaining the maximal benefit from this form of treatment.

Mayo Clinic, Rochester, Minn.

REFERENCES

1. DESJARDINS, A. U.: Quoted by Dysart.
2. DYSART, B. R.: X-ray Treatment of Infections: Review of the Literature and Report of Cases of Mastoiditis and Sinusitis. Ann. Otol., Rhin. & Laryng. **48:** 433–442, June 1939.
3. GATEWOOD, E. T.: Of What Value Is Roentgen Therapy for Sinusitis? A Rhinologic Study of Twenty-two Cases. Arch. Otolaryng. **31:** 275–282 February 1940.
4. KORNBLUM, KARL: The Present Status of Roentgen Therapy in Sinusitis. Ann. Otol., Rhin. & Laryng. **50:** 523–543, June 1941.
5. LARSELL, O., AND FENTON, R. A.: Quoted by Gatewood.
6. WILLIAMS, H. L., AND POPP, W. C.: Roentgen Therapy for Acute Sinusitis. Ann. Otol., Rhin. & Laryng. **49:** 749–754, September 1940.

Further Observations on the Radium Treatment of Postoperative Parotitis[1]

ROBERT E. FRICKE, M.D.,[2] and GORDON F. MADDING, M.D.[3]

Rochester, Minn.

ACUTE PAROTITIS has always been recognized as a serious condition. It has been the object of extensive research and much capable experimentation; many excellent statistical studies have been reported.

One of the first reports of acute parotitis appeared in the *Lancet* in 1829 (11). In this case the disease progressed to gangrene in spite of incision and drainage. In 1834 Brodie, in a clinical lecture, distinguished acute parotitis from epidemic parotitis, or mumps, and stressed the seriousness of the condition; he mentioned that when it appears as the termination of some other disease it usually indicates approaching dissolution. The disease was first accurately studied from a pathologic standpoint by Virchow, in 1858 (7).

Acute postoperative parotitis was recognized in 1878, when Munde reported a case following ovariotomy. In 1880 Moeriche reported that the disease occurred in 5 of 200 patients in whom ovariotomy was performed (2). The old well known association between the parotid and genital organs, as evidenced by the frequency of orchitis and mastitis as complications of epidemic parotitis, led to the assumption that postoperative parotitis was prone to follow operations on the pelvic organs (2). Paget reported cases of acute parotitis subsequent to injury or disease of the abdomen or pelvic organs. Many writers, however, have emphasized that of the earliest major surgical procedures the majority were operations on the ovaries (14). In later years, acute parotitis has been found to follow practically any surgical operation, especially in a septic field.

[1] Presented before the Radiological Society of North America, at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

[2] Section on Therapeutic Radiology, Mayo Clinic.

[3] Fellow in Surgery, Mayo Foundation.

ETIOLOGY

There have been much confusion and debate over the cause of acute postoperative parotitis. In the main, the arguments have had to do with whether the infection is hematogenous or lymphogenous or the result of an ascending infection by organisms present in the mouth. Much excellent experimental work and clinical observation have established the route last mentioned as the probable avenue of infection. Claisse and Dupré injected bacteria into Stenson's ducts in dogs but could not produce suppuration of the parotid gland unless the secretion of the gland was diminished by ligation of the ducts or by the introduction of foreign bodies. Berndt, Buck, and Buxton reported the results of injection of bacteria into Stenson's ducts in dogs and into the blood stream. A different pathologic picture was obtained after injection of the ducts than after injection into the blood stream; infection was accomplished with greater ease through the ducts and the histologic picture resembled that of acute parotitis of human beings. From the results of these experiments and from clinical observations, it is logical to assume that infection may readily travel up Stenson's duct from the mouth in cases in which salivary secretion has been seriously diminished. This diminution of secretion is common in debilitated patients following a serious operation. The administration of sedative drugs and the cessation of ingestion of food and fluids by mouth lessen the secretion and seem to favor the onset of acute parotitis.

DIAGNOSIS

There is no difficulty in the diagnosis of postoperative parotitis. The disease usually occurs two to fifteen days after operation, more often from the second to the

fourth day (8). Its incidence as a postoperative complication is low. It usually is said to occur in one of every thousand or every two thousand cases in which a surgical operation is performed. There is a "slight febrile excitement," as one of the old English writers (3) has put it, or the fever may be high (104 to 105° F.). The parotid gland is swollen and painful. There often is inability to open the mouth. In 20 per cent, or slightly more, of the cases the infection is bilateral. The staphylococcus is usually the offending organism in this country and in England and Germany (8). We have seen the submaxillary and sublingual glands involved, but much more rarely than the parotid gland. In contrast with the parotid gland, which is a serous gland, the other salivary glands are mucous glands, and mucus has been found a poor culture medium for bacteria. The infection either terminates by resolution or a large abscess may form. Fluctuation may eventually become apparent, although not invariably, as the thickness of the capsule of the gland may disguise it. Finally, spontaneous rupture may occur outwardly or through the ear or throat. The infection may eventuate in gangrene or in septicemia.

THERAPY

Early therapeutic efforts consisted of the application of leeches and poultices, the administration of laxatives by mouth, and incision and drainage when necessary. Leeches were mentioned with respect as late as 1906 in Osler's *Practice of Medicine* (9). These measures, together with a dependence on the strong constitution of the patient, sufficed until the value of irradiation in the treatment of acute inflammations was appreciated. The report of Rankin and Palmer, appearing in 1930, awakened new interest in the treatment of this condition.

The value of irradiation therapy has been stressed recently by Costolow, Mershon, and Johnson in an excellent report, in which they placed the mortality rate of this disease at 41 per cent in cases in which irradiation was not used but only 16 per cent when irradiation was used. Pearson also has stressed the value of irradiation.

In a series of cases of postoperative, parotitis seen over several years we used radium therapy in 58 per cent, having been guided by the following general principles: 1. Treatment is applied to the infected region as soon after diagnosis as possible (an emergency set-up is always ready for use). 2. The radium is taken to the patient's bedside and can be applied with a minimum of discomfort no matter how serious other complications may be. Often the treatment has been applied while oxygen was being administered. 3. A small sub-erythema dose has been found sufficient. The treatment usually limits the infection and averts the necessity of surgical incision and drainage. 4. Supplementary measures, such as oral hygiene and the application of moderate heat or of cold compresses, may be employed immediately following the termination of radium therapy.

Up to two or three years ago, the dosage was 50 mg. of radium sulphate (element) filtered with 1.5 mm. of monel metal and 2 mm. of lead at a distance of 1 inch (2.5 cm.) from the skin. Treatment was applied to each of four portals directly over the swollen parotid gland. Four balsa wood blocks, each 3 cm. square at the base, arranged in the form of a square, supported the radium tubes. Treatment was continued for eight hours, 896 roentgens being applied to the parotid region. A second application, with the same factors, was made to the periphery of the parotid region.

In the last few years the lead filter has been omitted entirely and the time shortened to six hours. This method yields 972 roentgens over the parotid. The same treatment is applied to the surrounding region. With this dosage the same result has been achieved and the weight over the tender infected gland has been decreased.

In clinics without adequate supplies of radium, but with portable equipment for roentgen therapy, moderate voltage (130

kv.), 4 or 6 mm. of aluminum filtration, depending on the thickness of the swelling, and a dosage of 50 to 75 roentgens may be employed. This treatment may be repeated in two days, and a third treatment may be given two days later, if needed.

PROGNOSIS AND RESULTS

In most of the excellent articles on the treatment of acute postoperative parotitis the authors seem to be unduly pessimistic. Charlton, in 1931, reported a mortality rate as high as 42.8 per cent, and in a study of the literature up to 1935, Green quoted a mortality rate of 25 to 60 per cent, the average in this country being 58 per cent. This figure has been widely accepted by subsequent authors. As Rankin and Palmer have emphasized, however, postoperative parotitis usually follows serious operations, especially those on the large intestine. It seems to us that the mortality rate in published reports has mainly been computed just as the mortality rate of cancer has been computed, that is, the death of any patient who had postoperative parotitis has been attributed directly to that complication. In our own experience, acute parotitis has not appeared to be a very fulminating or serious infection. To prove or disprove this theory, we have reviewed the records of 190 cases of postoperative parotitis observed at the Mayo Clinic from Jan. 1, 1927, to Dec. 31, 1940, inclusive. Table I shows the sites of the operations performed before the development of parotitis.

Radium therapy was employed in 111 cases and other measures (heat, cold compresses, gentian violet, compound solution of iodine, intravenous administration of mercurochrome, sulfanilamide, oral hygiene, diathermy, etc.) were relied upon in the remaining 79 cases.

In 19, or 10 per cent, of the 190 cases the patients died. Instead of ascribing this 10 per cent mortality to parotitis, we felt it worth while to analyze the protocols of the 19 cases to determine the rôle of parotitis in the final outcome.

As mentioned previously, 79, or 41.6 per cent, of the 190 patients had been treated by methods other than irradiation, and 111, or 58.4 per cent, had received radium therapy. Of the 79 patients who were not treated with irradiation, 6, or 7.6 per cent, died, while 13, or 11.7 per cent, of those who were treated with radium died. In so small a series, no significance can be attached to this distinction; radium treatment was often applied as a last resort in cases in which the patients were critically ill or in which other treatment had failed.

TABLE I: SITES OF OPERATIONS PERFORMED BEFORE THE DEVELOPMENT OF PAROTITIS

Site of Operation	In All Cases	In Fatal Cases
Colon	56 (29.5%)	7 (36.8%)
Pelvis	41 (21.6%)	1 (5.3%)
Biliary tract	31 (16.3%)	3 (15.8%)
Stomach	24 (12.6%)	3 (15.8%)
Miscellaneous	38 (20.0%)	5 (26.3%)
TOTAL	190	19

The ages of the patients and the type of operation performed before the development of parotitis are of interest, especially in the fatal cases. In the entire group of 190 cases the ages ranged from five to seventy-five years, with an average of 49.6 years. In the 19 cases in which death occurred, the ages ranged from thirty-seven to seventy-two years, the average being fifty-nine years, that is, ten years more than the average for the entire series. The sites of operation in the 19 cases in which death occurred are listed in Table I. In 9 of these cases operation was performed for a malignant growth. These findings indicate that parotitis which follows serious operations, such as those for malignant tumors or those involving the colon, stomach, or biliary tract, is more likely to prove fatal than is parotitis which occurs after less serious operations.

According to Rankin and Palmer, the prognosis is more serious in cases in which an abscess of the parotid gland has to be incised and drained than it is in cases in which such treatment is not necessary. This conclusion has been confirmed by other authors, and also holds true in our series of cases. Incision and drainage were

necessary in 24 of the entire series of 190 cases. In 3, or 12.5 per cent, of the cases in which this procedure was employed the patients died. In the 166 cases in which the parotid gland was not incised, the mortality rate was 9.6 per cent. In 7 of these 166 cases, an abscess of the parotid gland ruptured spontaneously, and all 7 patients recovered. In 2 of these cases drainage occurred through the ear and in the remaining cases through Stenson's duct.

Radium therapy appeared to have a definite influence on the percentage of cases in which incision and drainage were required. Whereas this procedure proved necessary in 15, or 19 per cent, of the 79 cases in which radium therapy was not employed, it was necessary in only 9, or 8 per cent, of the 111 cases treated by radium. Radium was used in 5 of the cases in which an abscess of the parotid gland ruptured spontaneously.

Sulfanilamide was used in 3 cases. These patients recovered, but in one case the administration of the drug had to be discontinued because of the development of severe cyanosis.

The causes of death in the 19 fatal cases are listed in Table II. Necropsy was performed in 7 cases: in 2 in which death was due to peritonitis, in 2 in which it was due to bronchopneumonia, in the 2 in which it was due to pulmonary embolism, and in the single case in which it resulted from staphylococcic septicemia.

TABLE II: MAIN CAUSES OF DEATH IN NINETEEN FATAL CASES OF POSTOPERATIVE PAROTITIS

Peritonitis	8 cases
Bronchopneumonia	4
Uremia	2
Pulmonary embolism	2
Cardiac collapse	1
Staphylococcic septicemia	1
Parotitis	1
TOTAL	19

We believe that acute parotitis was responsible for only one of the 19 deaths. In the other 18 cases, it played a very minor rôle. In fact, in studying the clinical reports of these 18 cases we found that in 2 the parotitis had completely cleared up, and in 3 additional cases the infection was subsiding satisfactorily at time of death.

In the case in which death was ascribed to parotitis, the infection followed cholecystectomy on a patient who had diabetes mellitus. Parotitis developed three days after the operation and proceeded to definite fluctuation in spite of prompt treatment with radium. Surgical incision and drainage were carried out on two occasions, but failed to produce any improvement, and death supervened.

SUMMARY AND CONCLUSIONS

Acute postoperative parotitis has serious potentialities. In fact, reports in the literature have been unduly pessimistic; Green's figure of 58 per cent as the average mortality rate in this country has been widely cited. Although acute postoperative parotitis is a serious complication, we do not believe that the prognosis is hopeless. We agree with Talbot, who said that when death occurs the primary disease process for which operation was performed is usually responsible and the parotitis is only a contributing factor. This was found to be true in the 190 cases which form the basis of this paper. In 111 cases the patients were treated with radium and in 79 by other methods, that is, approximately 60 per cent of the total number of patients received radium treatment and approximately 40 per cent were treated by other procedures. Approximately 11 per cent of the patients treated with radium later died and approximately 7 per cent of those not treated with irradiation died. This difference is not especially significant when selection of treatment is uncertain and based on many factors.

Spontaneous rupture and drainage occurred in 7 cases, but all of these patients recovered. In 8 per cent of the cases in which radium was employed and in 19 per cent of cases in which the patients were treated by other methods, the extent of the infection increased, fluctuation appeared, and surgical drainage became necessary. When incision and drainage are required, the prognosis is much more serious, according to most writers.

Of the entire group of 190 patients, 19, or 10 per cent, died. This latter figure would represent the mortality rate if the deaths were blamed entirely on the parotitis, as they frequently are in studies of this disease. A detailed study of the fatal cases, however, revealed that parotitis was the chief cause of death in only one case. The average age of the patients who died was ten years more than the average age of the entire group. In 9 of the 19 fatal cases the preceding operation had been performed for a malignant tumor. Operations on the colon were the most common surgical procedures in the fatal cases.

The principal cause of death in the majority of the 19 cases was peritonitis, with bronchopneumonia second, followed by uremia and pulmonary embolism. In 5 instances the parotitis had either cleared up entirely or was subsiding satisfactorily at the time of death.

Mayo Clinic, Rochester, Minn.

REFERENCES

1. BERNDT, A. L., BUCK, R., AND BUXTON, R. VON L.: Pathogenesis of Acute Suppurative Parotitis: An Experimental Study. Am. J. M. Sc. **182:** 639–649, November 1931.

2. BLAIR, V. P., AND PADGETT, E. C.: Pyogenic Infection of the Parotid Glands and Ducts. Arch. Surg. **7:** 1–36, July 1923.

3. BRODIE, B. C.: Inflammation of the Parotid Gland and Salivary Fistulae. Lancet. **1:** 450–452, Dec. 20, 1834.

4. CHARLTON, P. H.: Post-Operative Parotitis. Ann. Surg. **93:** 837–843, April 1931.

5. CLAISSE, PAUL, AND DUPRÉ, ERNEST: Etude anatomo-pathologique de quelques cas de parotidite. Bull. et mém. Soc. anat. de Paris. **69:** 32–38, January 1894.

6. COSTOLOW, W. E., MERSHON, H. F., AND JOHNSON, ROY: Treatment of Surgical Parotitis with Radium. Radiol. Rev. & Mississippi Valley M. J. **60:** 42, March 1938.

7. CUSTER, R. P.: Acute Suppurative Parotitis: A Pathologic and Bibliographic Study with Report of Two Cases. Am. J. M. Sc. **182:** 649–661, November 1931.

8. GREEN, M. T.: Postoperative Parotitis as a Complication of Abdominal Surgery. Tri-State M. J. **7:** 1406–1408, January 1935.

9. OSLER, WILLIAM: Principles and Practice of Medicine. New York, D. Appleton & Company, Ed. 6, 1905, p. 491.

10. PAGET, STEPHEN: Parotitis after Injury or Disease of the Abdomen or Pelvis. Brit. M. J. **1:** 332, Feb. 12, 1887.

11. Parotitis Terminating in Gangrene. Report of Hôtel Dieu. Lancet. **2:** 540–541, July 25, 1829.

12. PEARSON, E. A.: Parotitis and Staphylococcus aureus Septicemia as Surgical Complications. West. J. Surg. **47:** 468–473, August 1939.

13. RANKIN, F. W., AND PALMER, B. M.: Post-Operative Parotiditis: Treatment without and with Radium. Ann. Surg. **92:** 1007–1013, December, 1930.

14. TALBOT, H. S.: Acute Suppurative Parotitis: Its Etiology, Pathogenesis, and Treatment. Am. J. Surg. n.s. **25:** 267–276, August 1934.

The X-Ray Treatment of Acute Peritonitis[1]

JAMES F. KELLY, M.D., F.A.C.R., and D. ARNOLD DOWELL, M.D.

Omaha, Nebraska

THE TREATMENT of acute peritonitis at the bedside with x-ray was begun by us in 1934, and our early experience was reported before the section on Radiology of the American Medical Association in June 1938. In the last three years we have added several cases to the series presented at that time, and this new report, though leaving many questions unanswered, will give the reasons why we still believe in the procedure and think it worthy of more general adoption.

Since 1928 there has been no doubt in our minds that x-rays should be used with other conservative measures in the treatment of gas gangrene. That others agree with this conclusion is evidenced by the fact that over twenty favorable reports, covering more than 100 cases, now appear in the literature, showing that the mortality and morbidity in this disease have been definitely reduced when x-ray therapy has been used as an aid in its treatment. While adverse criticisms of the use of x-rays in gas gangrene have appeared since the advent of the sulfonamides, no series of cases adequately treated by irradiation has been introduced into the literature, with detailed data on specific cases, showing any high percentage of failures. For the honest practitioner who is a physician first and a specialist last, we believe that the status of the x-ray as an aid in the prevention and treatment of gas gangrene is settled. Other measures now undergoing trial may prove successful in the future, but what the x-ray can do is established.

Reference to the use of x-rays in the treatment of gas gangrene in association with a discussion on the treatment of acute peritonitis is not irrelevant. Few physicians are aware of the half century of controversy waged in the literature, and participated in by research workers and clinicians, concerning the exact rôle of *Cl. welchii* in the etiology of the most common form of acute peritonitis, namely, that following appendicitis, intestinal obstruction, and other lesions in which the peritoneal cavity becomes contaminated with intestinal contents or their numerous bacteria.

In this report only brief reference can be made to the possible etiologic relationship between gas gangrene and acute spreading peritonitis of intestinal origin. If they have the same causative organisms, our excellent results with x-ray therapy in the few cases here reported are explained. Regardless of the question of bacterial etiology, however, we are convinced that the results of irradiation in acute peritonitis will parallel those secured in gas gangrene and that a great reduction in the mortality following appendicitis will result.

HISTORICAL

Not many years after peritonitis was established as of infectious origin, Welch (1) and his colleagues published their first report on the anaerobic group of organisms. Shortly after this (1892–1896) Welch and Flexner (2) found *Cl. welchii* in severe cases of acute peritonitis. Veillon and Zuber (3) in 1898 stated that the anaerobes were the most important organisms in appendicitis and also reported that they had demonstrated an anaerobic coccus. Thus started the long procession of investigations into the bacteriology of peritonitis.

It is evident that before the turn of the century the claim that *Cl. welchii* was a factor in peritonitis had some supporters. Many others, however, even at that early date were more inclusive in their claims and, like Veillon and Zuber, stated that the infection was due to multiple organ-

[1] From the Department of Radiology, Creighton University School of Medicine. Presented before the Radiological Society of North America at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1-5, 1941.

isms, of which the anaerobic cocci, as well as bacilli, were present in appendicitis and some forms of peritonitis.

A prominent American investigator in this field is Bower (4) of Philadelphia. Like others he has reported a lower mortality in peritonitis cases treated with *Cl. welchii* serum than in cases which received no serum. That better results have not been obtained is probably due, as in gas bacillus infection, to the difficulty of neutralizing with any one stock antitoxin all the toxins produced by the several organisms present in this mixed infection.

From Altemeier's recent work (5) with the Ford Hospital group it is evident that peritonitis following appendicitis is usually a mixed infection, as a rule by three or more anaerobes growing in symbiosis. Altemeier did not demonstrate *Cl. welchii* as frequently as many workers claim it can be found, but he indicates in his excellent report the great difficulty of designating with absolute accuracy the rôle of each organism. In his long list of references to the literature are some reports minimizing the importance of *Cl. welchii*, but the great majority agree that a variety of anaerobes are present; with this latter conclusion Altemeier and his colleagues are in accord.

In our very early experience with gas gangrene we were impressed with the difficulty of drawing any fine conclusions about the exact rôle of the many organisms involved. In peritonitis we were impressed with the same difficulty. The bacteriology is too intricate and too complicated for present understanding and requires further investigation. The fact remains, nevertheless, that in some major aspects the two diseases are very similar: they are mixed infections; the anaerobes play an important part; the tissues involved often show gangrenous changes if the infection is allowed to progress, and the severe toxemia has been commented upon by all clinicians. Other similarities may be left for consideration elsewhere.

It would seem more important to appreciate the rôle played by the anaerobes as a group in gas gangrene and in acute peritonitis of intestinal origin than to indulge in a controversy as to how often a single one, namely, *Cl. welchii*, is the principal offender. This is particularly true when considering x-ray treatment, as radiation appears to have a beneficial effect upon any infection with this group of organisms. We are not much concerned, therefore, with the matter of specific organisms within the group, as this seems to be rather of academic than clinical importance. Suffice it to say that the more one studies these two diseases the more one realizes that etiologically, clinically, and to some extent pathologically, they show many identical features. The important similarity to keep in mind is that both respond to the same therapeutic procedure, namely, x-ray irradiation, and less promptly and less consistently to any other measure.

The value of the roentgen treatment of tuberculous peritonitis has been accepted for a quarter of a century or more and its importance in acute inflammatory pelvic conditions with peritonitis in the female has been quite widely recognized. There is, however, a definite scarcity of references in the American literature to this use of the x-ray; practically all the reports are from European sources, Fried (6, 7) being the outstanding contributor. Bertolotto's (8) conclusions are in agreement with those of Fried.

Our observations have been independent of any specific knowledge of the work of Fried and Bertolotto, but with the exception of some technical details they are in full accord with the conclusions of these investigators. They use a longer space factor and heavier filtration with fewer treatments than we have employed, but their clinical results are essentially the same. The earlier treatments are begun, the more prompt the results; treatments in the chronic stages are of less value. The lowered cost of care as a result of early treatment has also been noted by these writers.

We consider the short space factor quite

essential in the treatment of the fulminating toxic type of infection having a high mortality, such as gas gangrene, but this factor, as well as the r unit dose per treatment and total dose, is still subject to clinical experimentation and observation. Animal investigations such as those of Dowdy and Sewell (9) will undoubtedly play an important part in advancing the x-ray therapy of infections due to the anaerobes.

TYPES OF PERITONITIS

In our previous report, covering only a few cases, no attempt at a division into types or stages was made. However, at this time we have more cases, and though not enough for conclusive results, still enough to report the basis upon which we are working and to point out the trend which we expect the study to follow.

First of all, let it be understood that the treatment of all types of peritonitis with the technic advocated here is obviously out of reason. Tuberculous peritonitis and peritonitis due to fungi and numerous other types of organisms and irritants are not included in this discussion, which has special reference to the acute infection running a short course of one to four weeks and resulting in recovery or death. Time will permit us to discuss only the most common form encountered, namely, that secondary to appendicitis. The treatment of other types must be left for consideration elsewhere.

PERITONITIS SECONDARY TO APPENDICITIS

Before presentation of the treatment of any specific cases, it may be well to make it clear that x-rays are advocated only as an *aid* in the treatment of peritonitis. Surgery is the major therapeutic procedure for appendicitis, and irradiation is recommended solely as an agent to assist in localizing the infection and minimizing the toxemia. If the x-ray will accomplish these objectives, it will be in great demand by the surgeon.

When one speaks of appendicitis peritonitis he has in mind two areas of infection, one developing subsequent to the other. In our consideration of treatment, therefore, we shall divide the condition into stages, since the technical procedure applicable in the x-ray treatment at one stage may cause little or no benefit in another stage.

The following clinical stages of acute, spreading peritonitis secondary to appendicitis may be recognized:

A. Localized
   First stage: Appendicitis
   Second stage: Appendicitis plus localized peritonitis, which may go through all stages to abscess formation.
B. Generalized
   Third stage: Early spreading peritonitis; serosanguineous stage, with blood and serum in the peritoneal cavity and over the loops of bowel, with probably a small amount of pus. No adhesions, since the serum has not yet coagulated and formed fibrinous bands between the various intestinal loops.
   Fourth stage: Fibrinopurulent stage. Blood is disappearing; serum is becoming fibrinous and thicker and is forming bands between loops of bowels; some pus is accumulating.
   Fifth stage: Many adhesions are present which are more or less organized and there is a profuse collection of pus with probably some localized abscesses from time to time.

X-RAY TREATMENT

Since the pathological processes may vary considerably at the time x-ray therapy is requested, it seems well to discuss treatment in the various stages separately.

1. Appendicitis: Surgical removal is the treatment for acute appendicitis. The addition of some x-ray therapy preoperatively as a prophylaxis against peritonitis, or postoperatively if general peritonitis is feared, has no bearing on the necessity for the surgical removal of the appendix when it is acutely diseased.

2. Appendicitis with Localized Peritonitis: As in uncomplicated appendicitis, so in the patient who is operated upon

early and in whom the infection is confined to the adjacent peritoneum, x-ray therapy as a rule is not indicated. If, however, operation is not performed early, and, as is often the case, the patient is not seen until he has a walled-off abscess in the lower right quadrant, he should receive at least one x-ray treatment each day for three days after the abscess is drained, for fear the general peritoneal cavity has been infected. In this instance, the x-ray is used prophylactically. Several deaths occurred in our general peritonitis group as the result of extension of the infection after drainage of a local abscess.

3. Early Acute Spreading Peritonitis in the Serosanguineous State: Early spreading peritonitis was the stage in which our work with peritonitis was begun, and it is in this type of case that results are most spectacular. The response at this time is greatest because the infection has not produced such extensive local changes as to interfere with rapid recovery. The prompt relief from the evidences of toxemia in this stage is most impressive.

These patients constitute a group in which the mortality at the present time without x-ray therapy is in the neighborhood of 40 to 50 per cent. Treatment is indicated twice or three times the first day, twice the second and third days, and once each day thereafter until they are definitely out of danger. The x-ray therapy of gas gangrene has lowered the mortality in that disease to 10 per cent or less. All that has been said concerning early and frequent treatment of gas gangrene can be repeated here.

4. Generalized Peritonitis, Fibrinopurulent Stage: There is usually considerable toxemia present in this stage, and x-rays should be used to control this feature of the disease. The tendency to prolonged convalescence is increased with every additional day of delay of x-ray therapy. Some prompt recoveries have been witnessed, but the response is usually not so impressive and the chances of avoiding other complications are less than in the previous stages.

Two or three treatments each day are indicated until the toxemia is controlled. If evidences of toxemia reappear, treatment should be renewed and continued while these persist. Lower dosage is usually adequate under these conditions.

5. Generalized Peritonitis with Adhesions and Abscesses: In the stage of adhesions and abscesses there is not much chance for shortening the course of the disease with x-ray treatments, which are of little value when started so late. Indeed, x-ray therapy at this stage is not important unless the toxemia is still uncontrolled. When the disease is so far advanced, a long period of hospitalization is quite certain.

If secondary operations are necessary a dose or two of x-ray in the form of a two to three days' series may be required to localize the process and combat the toxemia.

If treatment is desired at this stage, two treatments daily for two or three days and others later as indicated may be of value in the exceptional case, but as a rule it is too late to shorten the course of the disease to any appreciable extent.

Clinical observation of the response to radiation in the early stages of gas infection or acute peritonitis would lead one to believe that the x-ray is an excellent aid to the patient who is toxic and attempting to establish a defense. After the patient has gained control of the situation and is less toxic, the effect of the x-ray is decidedly less evident. It is during the toxic invasive stages of these infections that the effect of the x-ray is more impressive, and it is during the early stages that irradiation should be used.

ANALYSIS OF RESULTS

The clinical impression we gained from our efforts in treating acute peritonitis in its various stages during the past few years was that we were accomplishing some real good, but to be certain about such matters all cases treated by various methods must be grouped for comparison and analyzed.

The statistical test applied here is based on the examination of all cases listed in the

record room of St. Joseph's Creighton Memorial Hospital as peritonitis and appendicitis treated in that institution from 1934 to 1940, inclusive. Charts of 3,579 patients were examined, of which 290 showed peritonitis in some form.

These 290 cases of peritonitis were divided into three main groups—localized peritonitis, local abscess, and generalized peritonitis.

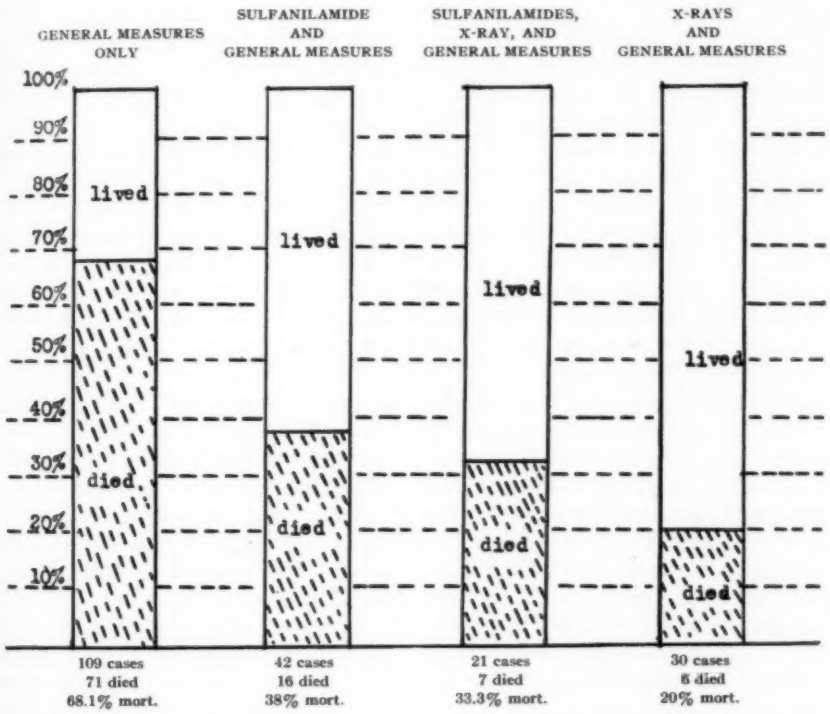


Fig. 1. Showing drop in mortality rate, with institution of x-ray therapy, in cases of generalized peritonitis secondary to appendicitis. The mortality in 51 cases (third and fourth columns) receiving x-ray therapy was 25.4 per cent; in 151 cases (columns 1 and 2) without x-ray therapy it was 57.6 per cent.

(1) There were 49 cases of peritonitis localized to the region of the appendix without abscess formation. All apparently remained localized, and the patients recovered regardless of what treatment they received in addition to surgery.

(2) There were 39 cases of peritonitis localized to the region of the appendix with abscess formation. Three patients died of pneumonia. The others recovered or developed generalized peritonitis and are considered under that heading.

In this series, therefore, all patients with uncomplicated appendicitis and all patients with only a localized peritonitis about the appendix without abscess formation recovered.

It is evident that the high incidence of recovery in the various stages of localized peritonitis makes such cases unsuitable material for determining the relative merits of various therapeutic methods. Surgery is supreme for the treatment of the local infection, and irradiation is apparently not indicated unless there is some danger of an extension of the process.

(3) Patients having generalized peritonitis with or without abscess formation numbered 202, with 100 deaths. The high mortality rate, 49.5 per cent, in this group

makes it quite suitable for a study of the different methods of treatment generally used in addition to surgery. The following combinations of methods appear to be of the most importance.

First, surgery supported by general measures but without x-ray therapy or the sulfonamides. General measures include the use of suction apparatus and other means of intestinal intubation, the oxygen tent, transfusions, etc. Of 109 patients thus treated, 71 died, a mortality of 68.1 per cent.

Second, sulfonamides and surgery with other general measures. Of 42 patients in this group, 16 died, a mortality of 38.0 per cent.

Third, a combination of x-rays, sulfonamides, and surgery with other general measures. Of 21 patients 7 died, a mortality of 33.3 per cent.

Fourth, x-ray irradiation as an aid to surgery and other general measures. There were 30 patients in this group, of whom 6 died, a mortality of 20 per cent.

We believe that the foregoing statistical data, though based on a relatively small number of cases, support our clinical impression that x-rays are of value in treating acute general peritonitis, and that when x-rays are used and the sulfonamides are omitted the result is better than with a combination of the two measures. It is also evident that the x-rays are much more efficient than the sulfonamides alone. We attribute the lower mortality in the combined sulfonamide and x-ray group as compared with the sulfonamide group alone to the fact that since the early part of 1938 we have refused to treat with x-ray unless the sulfonamides were stopped, usually from twelve to twenty-four hours before irradiation was begun. The x-rays were thus of assistance to those patients who had not received too much of the sulfonamides, and the lower mortality rate among this group is evidence of this fact. We believe that if both measures had been used simultaneously the mortality rate would have been higher than in the group treated by sulfonamides alone.

If all patients receiving x-ray therapy are considered together, we have a group of 51, with 13 dead, or a mortality rate of 25.4 per cent; if all receiving sulfonamides are grouped together, we have 63 cases, with 23 deaths, or a mortality of 36.5 per cent.

In our experience the best method of treatment is use of the x-ray as an aid to surgery and all the other methods of treatment usually employed with the exception of the early sulfonamides. Whether or not sulfathiozal is antagonistic has not been determined, but it is definitely not as inhibitory to the action of the x-rays as were the earlier sulfonamides. We arrived at this conclusion following the observation that some very sick patients who had been on sulfathiozal but who were obviously not responding in a satisfactory manner did respond to irradiation. Lately we have cautiously used the two measures simultaneously in some less seriously sick patients and as yet have not observed the antagonistic action so evident with the early sulfonamides. Whether there is a complete absence of antagonistic action, however, when sulfathiozal is used, or whether this is only a matter of degree, we are not able to judge. We do feel quite certain that we have been much more successful with it than with the other sulfonamides.

We have had only limited experience treating patients who have had any of the sulfonamides used locally in the peritoneal cavity. Our conclusions are based on the use of the early sulfonamides internally.

It seems superfluous to state that we are absolutely opposed to the trial of the sulfonamides for a few days with a subsequent changing to x-ray therapy, with or without the drug. A brief consideration of the changes in the underlying pathology as it progresses rapidly through its various stages should make it evident that during the time the sulfonamides are used the disease is reaching a stage less responsive to radiation therapy. Though the patient may live with the late assistance of the x-ray, the chances for a prompt recovery

with a minimum loss of time are very limited. In addition, he has the expense due to prolonged stay in the hospital. The lowered mortality and morbidity in acute peritonitis as in gas gangrene, as a result of x-ray therapy, make the use of this method of treatment imperative before gangrenous changes and other complications are established.

The value of early irradiation in peritonitis secondary to appendicitis cannot be overemphasized. When x-ray treatment is given early, during the acute spreading serosanguineous phase, the disease responds more promptly and more completely, seeming to undergo thorough resolution without complications. When treatment is started late, during the stage of plastic peritonitis with pus formation, there has been much more difficulty in controlling the situation as regards a prompt recovery. Convalescence is invariably prolonged and is associated with the discharge of large amounts of pus and with pocket formation. Repeated surgical interference may be necessary before the patient is safely convalescent.

The x-ray as a prophylactic will not be discussed at this time, but we have observed what we consider excellent results with its use and recommend it to prevent an extension of a localized peritonitis or to prevent the inception of peritonitis after injuries such as gunshot wounds.

The use of the suction apparatus has increased the necessity for early and correct diagnosis of acute intra-abdominal conditions. The use of the x-ray as a means of controlling the infectious element in acute peritonitis has added to this necessity.

Conditions which preclude the possibility of success in the treatment of peritonitis with x-ray are:

1. Delayed use of the x-rays—pathology too far advanced.
2. Organic obstruction of the bowel—not operated.
3. Open and leaking perforation in the bowel.
4. Internal use of the early sulfonamides simultaneous with x-ray therapy.
5. Underlying organic pathology in itself fatal, such as advanced cancer and its complications.

X-RAY TECHNIC

The x-ray technic cannot be definitely fixed, as the requirements for each case must be met promptly and individually to give the best results. Variables are the number of treatments given each day, one to three; r units per dose, 50 to 75, with occasionally less and sometimes more; kilovoltage, 90 to 130 kv.; filter, between 2 and 5 mm. of aluminum; distance, 40 to 50 cm. The clinical aspects of the case requiring certain factors from the above variables are not within the scope of this paper.

It goes without saying that young girls and pregnant women should be given x-ray treatments over the abdomen and pelvis with caution, and large doses should be avoided unless the condition is critical.

SUMMARY

1. A further report on the results of x-ray treatment of acute peritonitis of intestinal origin is made.

2. The possibility of a similar etiology for acute peritonitis of intestinal origin and gas bacillus infection is mentioned.

3. The underlying pathology of the various stages of this type of peritonitis are reviewed in a plea for the early use of x-ray therapy, before the condition is so far advanced that the best results are impossible of attainment.

4. The technical procedure is outlined in a general way. No specific technical directions are practical, since variations in age, weight, and sex, as well as the stage of the disease at the time treatment is given, make each case an individual clinical problem.

5. The mortality when x-ray therapy is used is only one-half what it is when the sulfonamides are used internally, and less than one-third of that with surgery and other general measures without either x-ray or the sulfonamides.

816 Medical Arts Building, Omaha, Neb.

REFERENCES

1. WELCH, W. H.: Morbid Conditions Caused by Bacillus aërogenes capsulatus. Bull. Johns Hopkins Hospital **11:** 185–204, September 1900.

2. WELCH, W. H., AND FLEXNER, S.: Observations Concerning Bacillus aërogenes capsulatus. J. Exper. Med. **1:** 15–45, January 1896.

3. VEILLON, A., AND ZUBER, A.: Recherches sur quelques microbes strictement anaérobies et leur rôle en pathologie. Arch de méd. expér. et d'anat. path., Paris **10:** 517–545, 1898.

4. BOWER, J. O.: Spreading Peritonitis Complicating Acute Perforated Appendicitis. J. A. M. A. **112:** 11–17, Jan. 7, 1939.

5. ALTEMEIER, W. A.: Bacterial Flora of Acute Perforated Appendicitis with Peritonitis. Ann. Surg. **107:** 517–528, April 1938.

6. FRIED, CARL: Roentgen Irradiation of Acute and Subacute Purulent Inflammations. Acta radiol. **9:** 109–116, 1928. Abst. in Am. J. Roentgenol. **21:** 301, March 1929.

7. FRIED, CARL: Roentgen Treatment of Inflammatory Pelvic Disease in Gynecology. Strahlentherapie **19:** 649–668, 1925. Abst. Am. J. Roentgenol. **14:** 471, November 1925.

8. BERTOLOTTO, UGO: Roentgen Therapy of Gynecological Inflammations. Radiol. med. **21:** 1103–1126, October 1934. Abst. in Am. J. Roentgenol. **35:** 134, January 1936.

9. DOWDY, ANDREW H., AND SEWELL, ROBERT L.: Roentgen Radiation in Experimental Clostridium welchii Infection (Gas Gangrene) in Dogs: Preliminary Report. Radiology **37:** 440–442, October 1941.

Acute Postoperative Parotitis[1]

EUGENE P. PENDERGRASS, M.D., and PHILIP I. HODES, M.D.

Philadelphia, Penna.

THE PURPOSE OF the present communication is to describe a method for treating acute postoperative parotitis radiotherapeutically which has produced very satisfactory results. Attention will also be directed to the importance of the supportive procedures which are necessary to assure free drainage from the infected gland into the mouth.

ETIOLOGY

Why acute pyogenic infections develop in parotid glands postoperatively remains a moot question, but considerable evidence seems to indicate that acute postoperative parotitis is the result of an ascending infection of Stenson's duct (2, 6, 7). Numerous theories have been postulated. Direct trauma to the parotid during operation has been suggested (5, 6). Against this concept is the observation that in over half of the cases reported spinal anesthesia has been used. Some men believe acute postoperative parotitis is the result of hematogenous invasion of the gland. We are informed that the organisms recovered from an infected parotid oftentimes do not correspond to the bacteria found in infections elsewhere in the body (2). Many surgeons, nevertheless, inspect the abdominal wounds carefully for a hidden wound abscess in those patients who develop acute parotitis.

In view of the frequency with which primary acute pyogenic infections of the parotid have occurred following gastrointestinal operations, it has been suggested that a vitamin A deficiency may act as a predisposing factor. Atrophy of the epithelial lining cells of the salivary glands has been reported as occurring in some instances in which there has been a lack of this vitamin (5). Parenchymatous degeneration of the parotid due to fever, and toxins from the primary disease have also been suggested as possible etiologic factors.

PATHOGENIC CONSIDERATIONS

Under normal conditions staphylococci may be found in the majority of "clean" mouths. They occur in varying concentrations in different portions of the mouth, being much more numerous around the parotid duct than in the sublingual region. The relative sterility of the sublingual region may be attributed to the incessant activity of the tongue, which keeps this area clean, and the fact that the secretions of the submaxillary gland are rich in mucin, which is absent in the parotid secretions (5). It is believed that mucin exerts an inhibitory effect upon staphylococci which is roughly proportional to its concentration (11, 12). This may account for the relative rarity of acute infections in submaxillary glands.

Patients with poor oral hygiene have much higher concentrations of oral bacteria than do patients with "clean" mouths (5). The greater susceptibility of the former group to parotid infections may be attributed to this difference in oral sanitation. It is also noteworthy that oral bacterial estimations made before and after operation reveal precipitous increases in bacterial concentration following surgery (10, 11). This is probably related to the patient's fluid balance, as the greatest changes are observed in patients with dry mouths, whereas patients with no oral dryness show little difference. Dehydration, vomiting, restriction of fluids, and decreased salivary secretion, which accompanies some surgical procedures and abdominal operations in particular, may therefore be a significant and predisposing

[1] From the Department of Radiology of the Hospital of the University of Pennsylvania, Philadelphia, Penna. Presented before the Radiological Society of North America at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

factor in the development of acute parotitis.

Histologically, acute pyogenic infections of the parotid start as a catarrhal inflammation of the larger ducts (6, 10). Within these ducts bacteria may be seen (6). The ducts become distended with retained purulent secretions, most marked in the larger ducts and least extensive in the smaller ones. Later the epithelium of the ducts becomes eroded. Periductal infiltration and necrosis take place where the duct-walls are broken, allowing penetration of the infection into glandular tissue (2, 6). The glandular tissue, however, is not always involved. This depends upon the virulence of the infection, the inherent protective mechanism of the host, and the presence or absence of ductal obstruction. When the glandular elements do become involved, multiple small abscesses develop, which gradually become larger and coalesce to form larger ones.

THERAPEUTIC CONSIDERATIONS

We consider acute parotitis a radiotherapeutic emergency. Irradiation is instituted as soon as the diagnosis is made. When the patient is first seen, an attempt is made to determine whether or not Stenson's duct is patent. This is done by gently massaging the parotid duct in a forward direction along its course. If a block exists, an attempt is made to remove the obstruction by aspirating and probing the duct. Whalebone filiforms (8) or a blunt-edged 18 or 20 gauge needle may be employed for this purpose. By careful probing of the duct in this manner, the obstruction is usually overcome. Occasionally gentle irrigation of the duct by means of a syringe attached to the blunt-edged needle may be necessary. When these procedures fail, the end of a suction apparatus is applied over the mouth of the parotid duct and strong suction is exerted. Not infrequently it is necessary to alternate probing and aspirating before adequate flow of secretions is obtained from the gland. The importance of establishing free drainage from the parotid cannot be overemphasized. Unless this is done, the best interests of the patient are not served.

After drainage from the duct has been established, the parotid is irradiated. As a rule we use roentgen rays generated at 130 kv., filtered through 0.25 mm. of copper + 1 mm. aluminum. The target skin distance is usually 25 to 30 cm., the portal large enough to extend beyond the confines of the inflamed area. Occasionally patients are too sick to be brought to the Radiological Department for treatment. In such instances, an ordinary diagnostic portable x-ray machine is used.

On the first day a dose of 150 r is delivered to the skin overlying the infected gland.[2] The patient is warned that the pain may increase following the exposure. On the second day, the parotid infection usually looks worse. At this time a second exposure of 150 r is given the infected gland. By the third day the patient usually feels better and a third exposure of about 100 r is administered. Usually no treatment is necessary on the fourth day. Occasionally on the fifth day the parotid is again treated, with 75 to 100 r. From the time the patient is first seen, every effort is made to promote emptying of the parotid.

The patient's fluid balance is watched carefully. He is encouraged and indeed forced to chew gum constantly and suck on hard sour candy. Hot mouth washes are employed at half-hour to one-hour intervals. Sour beverages stimulate parotid secretions and their use is encouraged. These methods for stimulating salivary secretion and maintaining oral cleanliness are rigidly enforced.

The character of the parotid secretion changes during the course of treatment. When first observed, it is usually thick and gummy. Parotid secretions seen in the first few hours of the disease are watery and may contain small flecks of débris. Only a few drops may be expressed. On the second and third days following the institution of roentgen therapy, the secretion is more viscid and more copious. It

[2] "r" in air, without back-scatter.

has the appearance of heavy cream and may vary in color from milky white to a somewhat greenish hue. With time, the excretion gradually changes until it finally becomes of a watery consistency.

The rate of secretion of parotid saliva has been variously established to be from 0.02 c.c. per minute to approximately 0.5 c.c. per minute (3, 4). Under unusual conditions, the secretion may reach 2.4 c.c. per minute. The rate of secretion being slow, the ductal system remains in constant danger of being occluded. It is therefore necessary to stimulate the parotid repeatedly and attempt to empty it by mechanical means. Such stimulation is maintained by chewing gum, sucking candy, and other procedures outlined above. Mechanical drainage depends upon the massaging action of the overlying jaw muscles and gentle manual massage.

Massaging the gland is an extremely important though somewhat hazardous procedure. The pressure exerted by the massaging fingers must be gentle. The parotid must be handled gingerly and with the care accorded other infections on the face. The gland is always "milked" in the direction of the normal flow of its secretions. Patients almost invariably complain of pain during the procedure. This should not deter one, as it is essential that drainage be maintained. The nurse and the patient are carefully instructed in this "milking" procedure. Thus, constant emptying of the parotid secretions and pus is assured throughout the day and night.

Lugol's solution, minims xx every three hours, has been advised for acute parotitis on the theory that the drug has an antiseptic value which it may exert while being secreted from the salivary glands (9). We have no opinion concerning this form of treatment. The few patients in whom we used Lugol's solution also received roentgen therapy and supportive care. The ethyl ether of β-methylcholine may prove of value. This drug increases salivary flow about ten-fold, the effect lasting several minutes (4). We have never used the ethyl ether of β-methylcholine but intend doing so in our next patients with acute postoperative parotitis.

We have treated 47 patients with acute postoperative parotitis with roentgen therapy. Of this group 19 (40 per cent) responded rapidly within a period of four days. In 13 patients (28 per cent) the improvement was less dramatic, though the course of the disease was somewhat shortened. Thus, 32 of the entire group of 47 patients (68 per cent) were benefited. Fifteen patients (32 per cent) had little or no effect from the treatment. In 7 instances incision and drainage of the gland were resorted to.

Death from parotitis alone is rare. It is, therefore, extremely difficult to determine the mortality attending this complication. Whereas in some individuals parotitis is the chief factor responsible for death, there are others in whom the mortality must be attributed solely to the primary disease. Our data suggest that acute postoperative parotitis may have been the precipitating cause for death in from 20 to 30 per cent of the cases.

It is only fair to state that the results reported above represent the findings in the entire group and include many patients who did not receive the comprehensive supportive care that now is regarded as essential. Our recent results, in patients in whom adequate parotid drainage has been maintained, have been much more satisfactory. Six patients belong in the latter group. All are well. One required incision and drainage.

CONCLUSIONS

Roentgen therapy is a valuable adjunct in the treatment of postoperative parotitis and will cure most patients if meticulous supportive care is provided and oral cleanliness and constant parotid drainage are maintained.

3400 Spruce St., Philadelphia, Penna.

BIBLIOGRAPHY

1. ALLEN, F.: Secretory Activity of the Parotid Gland. Quart. J. Exper. Physiol. 19: 337–362, July 1929.

2. BERNDT, A. L., BUCK, R., AND BUXTON, R. VON L.: Pathogenesis of Acute Suppurative Parotitis: Experimental Study. Am. J. M. Sc. **182:** 639–649, November 1931.

3. BRAMKAMP, R. G.: Urea and Chlorides in Human Parotid Saliva; Changes at Different Rates of Secretion as Affected by Atropine and Pilocarpine. J. Lab. & Clin. Med. **22:** 677–680, March 1937.

4. COMROE, J. H., JR., AND STARR, ISAAC, JR.: Further Studies on the Pharmacology of Acetyl-β-Methylcholine and the Ethyl Ether of β-Methylcholine. J. Pharm. & Exper. Therap. **40:** 283–299, November 1933.

5. CRILE, G., JR., AND MANNING, W. R.: Postoperative Parotitis. Am. J. Surg. **50:** 664–667, December 1940.

6. CUSTER, R. P.: Acute Suppurative Parotitis: Pathologic and Bibliographic Study with Report of 2 Cases. Am. J. M. Sc. **182:** 649–661, November 1931.

7. HOBBS, W. H., AND SNEIERSON, H.: Infections of Parotid Gland: Further Studies on Etiology and Treatment; Sialograms of Normal and Abnormal Glands and Ducts Including Tumors. Am. J. Surg. **32:** 258–271, May 1936.

8. HOBBS, W. H., SNEIERSON, H., AND FAUST, C. L.: Acute and Chronic Infections of Parotid Gland: Treatment by Dilatation of Stenson's Duct. Surg., Gynec. & Obst. **54:** 555–563, March 1932.

9. LEITHAUSER, D. J., AND CANTOR, M. O.: Lugol's Solution in Acute Secondary Parotitis. Ann. Surg. **101:** 1171–1174, May 1935.

10. PAYNE, R. T.: Infection of Salivary Glands. Proc. Roy. Soc. Med. **31:** 398–417, February 1938.

11. SIEFERT, E.: Ueber den Infektionsweg bei postoperative Parotitis. Deutsche Ztschr. f. Chir. **198:** 387, 1926.

12. TALBOT, H. S.: Acute Suppurative Parotitis: Its Etiology, Pathogenesis and Treatment. Am. J. Surg. **25:** 267–276, August 1934.

DISCUSSION OF SYMPOSIUM ON INFLAMMATIONS

(Papers by A. U. Desjardins; J. P. Rousseau *et al.*; W. C. Popp and H. L. Williams; R. E. Fricke and G. F. Madding; J. F. Kelly and D. A. Dowell; E. P. Pendergrass and P. J. Hodes)

H. Dabney Kerr, M.D. (Iowa City, Iowa): Dr. Pendergrass is to be congratulated on his able arrangement of the symposium which again brings to the foreground, as it should, the vital question of irradiation of inflammatory conditions.

It is a privilege as well as a responsibility to be asked to open the discussion on this group of interesting papers. Dr. Desjardins has given us in his usual clear and convincing style his concept of the basic action of radiation on inflammatory conditions. He also cites good experimental evidence in support of his contention that the effect is chiefly on the polymorphonuclear leukocytes and lymphocytes. That these cells are much more sensitive than most other tissues of the body, I think no one denies, and their destruction may well be the basic reason for the favorable response of inflammations to irradiation. I am personally inclined, however, to the view that it is not the only factor, and that increased blood supply may play a considerable rôle. I have seen a local temporary erythema due to vascular dilatation occur in the skin within a half hour and most of us, I am sure, have observed rapid acute swelling of the neck after moderate doses to nodes in this region. These changes must, I think, be explained on the basis of vascular phenomena and not cellular disintegration *per se*. I believe, also, that there may be some unrecognized factors which have a particular bearing on the relief of pain.

I should like to ask Dr. Desjardins to what he attributes the favorable response of inflammations which are apparently of toxic and not bacterial origin, and of those lesions in which there is calcium deposit, as so-called bursitis or peritendinitis. In some of these the calcium disappears quite promptly following small amounts of radiation. I wonder if this can be explained on the theory of cellular destruction. I find it difficult to explain on the basis of hyperemia alone, since many cases that respond to irradiation have failed to benefit from previous physical therapy in the form of diathermy or other heat-producing measures. The question of pain relief is also connected with this problem.

Dr. Rousseau has made a valuable contribution to our knowledge of the treatment of pneumonia. He rightly assigns to the sulfonamides the first place in treatment but brings forcibly to our attention the necessity of remembering that in those cases which do not respond favorably to chemotherapy and in those in which chemotherapy is contraindicated, we have a second line of defense in the roentgen ray. I think many of us have ceased to think of pneumonia as a disease in which radiation plays any significant therapeutic rôle, but Dr. Rousseau has shown this to be a mistake.

I was much interested to hear that Dr. Rousseau makes sure that the sulfonamide is discontinued before irradiation, as we have gained a very definite clinical impression that the combined effect of these drugs and roentgen rays is not so good as that of either agent used alone. This clinical impression we have confirmed experimentally in mice infected with staphylococcus, using sulfanilamide. Either measure was fairly efficacious when used alone, but when both were used the organisms were much more lethal. The mechanism of this interaction is not at all clear. It may have some relation to the breaking down of sulfanilamide, a phenomenon which occurs when it is irradiated with ultraviolet rays. According to Fox and his collaborators, a variety of unstable substances in various stages of oxidation and reduction are formed when sulfanilamide is irradiated, but the bactericidal properties of these substances have not yet been worked out. As yet, however, there is no experimental evidence to show that roentgen rays cause the drug to break down.

I should like to ask Dr. Rousseau whether or not he has ever had evidence that any of his patients was sensitized to irradiation by the sulfonamide or *vice versa*. There are several reports in the literature

stressing this side of the picture. One patient who was receiving sulfanilamide for tonsillitis and cervical adenitis was apparently made much worse by the administration of two doses of 100 roentgens to the affected nodes. Regardless of these theoretical considerations, the fact remains that Dr. Rousseau was able to save a goodly percentage of his pneumonia patients in the face of contraindications to chemotherapy.

Dr. Popp and Dr. Williams have presented us with an interesting series of cases of sinusitis from which the usual objection of the otolaryngologist has been removed, namely, that the cases were studied and observed only by an enthusiastic radiologist. While that objection is undoubtedly valid in some instances, I think radiologists are no more prone to exaggeration than are other specialists. The co-authorship of a radiologist and otolaryngologist, however, adds to the value of the contribution. I think that more sinus disease should be treated by irradiation, and preferably in the early stages, but this will not occur until we have the cooperation of the other specialty and have broken down the feeling that exists among a great many that irradiation constitutes an infringement of their inalienable rights. I do not advocate not treating these patients by local means as well, but I insist that anything that will ease their pain and relieve the engorgement is worth while. Incidentally, the relief of engorgement would not appear to indicate that there is an increase in blood supply in these cases. In our experience, however, some prompt but transitory exacerbation of pain results before clearing occurs, and this, I think, does mean increased circulation.

May I ask Dr. Popp for his explanation of the phenomenon of the prevention of recurrent attacks of sinusitis? We have observed this prevention of recurrence in cases of localized herpes but we have not noted it in our sinus irradiation, although, frankly, we do not have a large volume of this type of work. It is well worth noting that the results reported by Dr. Popp and Dr. Williams indicate that the treatment of sinusitis should not be too localized, but should include all sinuses and their ostia.

There is no question but that anything which will reduce the mortality or morbidity of peritonitis should be a great boon to the surgeon. It has been interesting to hear of Dr. Kelly's results in the treatment of this condition and he is to be congratulated on saving so many of his cases. I wonder if he will be kind enough to theorize on the probable mechanism of prophylactic irradiation. Is it an increased blood supply, a devitalization of the potentially present organisms, or the increase in antibodies which overwhelm the infection before it has a chance to spread?

I am interested in Dr. Kelly's statement that he suspects that sulfathiazole may not be contraindicated with irradiation. Our work was done only with sulfanilamide and we have insisted on withdrawing the drug at least 48 hours before irradiation is instituted. Perhaps we have been too cautious in this respect since the advent of the newer sulfonamides. Some experimental work along this line would be interesting and important.

Drs. Fricke and Madding and Pendergrass and Hodes have done us a service in again calling attention to the irradiation treatment of postoperative parotitis. Radium and roentgen therapy each has its advantages, but where it is feasible to move the patient I prefer the latter because the irradiation takes less time, is more uniform in its distribution, and does not necessitate placing an applicator on a very tender part. In the treatment of the bedfast patient, however, radium is very valuable.

I was especially pleased that in their presentation, Drs. Pendergrass and Hodes did not confine their remarks to irradiation treatment, as called for by the title of their paper, but emphasized ancillary forms of therapy directed at the primary cause of the infection. Too many of us are willing to irradiate patients sent to us without looking at the broader aspects of their disease. It behooves us, I think, to be constantly aware that first of all we are physicians, that all aspects of the patient's disease are of interest, and that neglect of any one phase may turn the tide against us.

Eugene P. Pendergrass, M.D.: Doctor Desjardins, will you comment on the work of Tuttle at the New York Hospital, who found that treated animals were more susceptible to induced skin infections than untreated ones?

A. U. Desjardins, M.D.: If I understand Dr. Kerr's question correctly, it is how I explain the effect of x-rays on inflammations or infections in which leukocytic infiltration is not a prominent feature. In inflammations of this character, or of any other character, it must be remembered that, besides leukocytic infiltration, hyperemia is taking place at the same time; in fact, the hyperemia begins before the infiltration. Under these conditions, the circulating blood in the inflamed region contains more leukocytes than it does under normal conditions. The rays act not only on the leukocytes which have migrated into the tissues, but also on those in the blood which is circulating in the vessels of the inflamed territory. In a condition such as chronic bronchial asthma, roentgen treatment may be directed toward the spleen, the liver, the mediastinum, the head, or, indeed, toward any part of the body through which a large quantity of blood circulates, and the effect on the asthma will be very nearly the same, regardless of the region treated. This is probably because the rays act primarily on the leukocytes in the circulating blood; a proportion of the more sensitive forms of leukocytes are destroyed, and the products of their disintegration are mixed with the blood stream; the result appears to

be essentially an autogenous protein reaction. Referring to inflammations in which leukocytic infiltration is a minor factor, as I explained in the paper, the fact that leukocytic infiltration is slight or practically absent may explain why small doses repeated at short intervals give better results than larger, single doses which are most effective in other forms of inflammation.

In connection with the second point raised by Dr. Kerr, concerning the action of roentgen rays on calcifying myositis or bursitis, there is no doubt that roentgen treatment often produces a definitely and rapidly favorable effect. Just how this effect is brought about has not yet been made clear. No doubt, a number of factors play a part, but their nature is still unknown.

Regarding the paper on sinusitis, which I read for the authors (Williams and Popp), the question was asked how roentgen treatment prevents the recurrence of such inflammations. Dr. Popp and I have talked about this many times, but we are not in a position to explain it.

As for the work of Tuttle and his co-workers, I cannot discuss this properly because of insufficient information about the technical details of irradiation. It is possible that the doses used might explain the development of septicemia. As you know, when inflammatory lesions are treated with roentgen rays, it is essential to get away entirely from the conception of tumor doses, since doses of such magnitude may not only increase the inflammatory process rather than diminish it, but may even produce sarcoma, as the work of Lacassagne would seem to indicate. Although evidence of this kind in human beings has never been presented, the fact of its occurrence in animals should be sufficient to make us pause.

With reference to the size of the field, my experience has been that treatment should be given widely beyond any visible involvement. When, for example, the inflammation appears to be confined to one cheek, the whole side of the head and upper half of the neck should be treated. When the inflammation appears to consist of a patch on the thorax, the whole front of the thorax down to the navel should be treated. The wider the area treated, within reasonable limits, the better the results. This is certainly true in conditions such as erysipelas or gas bacillus infection, probably because what is seen may be only a part of the process. Beyond the zone of involvement visible on the surface, there may be an internal zone of tissue in which the process is extending.

James P. Rousseau, M.D.: Dr. Kerr's question as to whether or not the previous use of sulfonamides may have sensitized some patients to x-ray, I am unable to answer. Most of the deaths in our small series of treated cases were so early that we were unable to attribute them to anything other than the severe pneumonic illness.

The question in regard to the combined use of sulfonamide therapy and roentgen therapy in particular infections has, I believe, already been answered by Dr. Kerr in his excellent discussion. It is certainly my feeling that until more is known about the reactions of both the sulfonamides and x-ray that the two should not be combined.

James F. Kelly, M.D.: The question, already discussed, as to whether the effect of the x-rays is on the blood vessels or on the cells, cannot be definitely answered. If, however, treatments are given with small doses at short intervals, no initial swelling occurs, the area of inflammation tends to regress, and there is, I believe, an increased blood supply, which means opening of the finer capillaries and small vessels, as Dr. Pendergrass has stated. I also feel quite certain, from the clinical aspects, that there is some antitoxic effect, which may be attributed to the increased production of antibodies by the cells. I see no reason why these two effects cannot occur simultaneously and without any conflict.

The question has been asked, how large a dose of x-ray would you give a young woman with peritonitis? In practically all of our anti-inflammatory treatments less than an erythema dose is given, and this in a period of two to five days. In a severe peritonitis our chief interest is in the patient's recovery, regardless of the possibility of later complications. Therefore, we might go a little above the usual 60 or 70 per cent dose, though as a rule this is not necessary. In the presence of a strangulated hernia or a perforation in the bowel which has not been repaired there will be continual contamination of the peritoneum and the continuation of the toxemia requires a prolonged series of x-ray treatments. The average patient receives about 60 or 70 per cent of a skin erythema, and with the low voltage and low filtration used, no harm should come to any young woman. We are especially careful about young girls and pregnant women.

Although Fried made his first report on the x-ray treatment of pelvic peritonitis in 1924, we have seldom been called upon to treat this condition, as recovery usually ensues without x-ray therapy. If pelvic peritonitis becomes a general peritonitis and the patient's life is endangered, we are then called upon; in such cases we treat the entire peritoneal cavity, and have obtained good results.

We believe that the use of x-rays for the prevention of peritonitis, after an operation on the bowel for instance, is extremely helpful. We do not hesitate to give 75 r units over the entire abdominal cavity once each day for three days, which is about the same dose that would be given to prevent osteomyelitis in a compound fracture.

Robert Fricke, M.D.: The questions which I am asked to consider make up in quality what they lack in quantity. One is: "Does x-ray therapy interfere with typing of sputum as do sulfa-drugs?"

I do not think so. I think that the treatment slowly and gradually increases the resistance of the tissues and that the infection regresses instead of continuing and forming an abscess, as is usually the rule. With the small dosage employed, the treatment naturally has no direct lethal effect on the bacteria themselves.

The next question is: "Do you often find that following irradiation for acute parotitis, the patient's general condition improves rapidly, but the enlarged gland requires something more in order to regress to its normal size?" It is true that the effect is gradual and one has to be patient, since it takes several days after treatment for the parotitis to regress. We have had to repeat treatments in only a small percentage of cases, and in these instances we have usually waited a week or ten days to make sure that another treatment was necessary. Often there is a delayed reaction—perhaps five or six days later the parotitis suddenly subsides.

A. U. Desjardins, M.D.: I am asked a question involving the dose of rays given at each session of treatment and the direction of the beams of rays in the treatment of acute sinusitis. The dose given to each field the first time is 75 to 100 roentgens. As I mentioned in my paper, the treatment is repeated, if necessary, two or three days later. The beams of rays should converge from each side of the face at an angle of 30 or 35 degrees, depending on the patient. Each half of the face should be irradiated separately, and the eyes and the bridge of the nose should be included.

A second question is: "Are you worried about the late development of cataract when the eyes are not protected during treatment?" If the dose is kept below the tolerance of the eyelids, there need be no fear of cataract. As you know, the eyelids are slightly more sensitive than the surrounding skin of the face, just as the skin of the scrotum is more sensitive than the skin of the leg. If the tolerance of the eyelids is borne in mind, it must be obvious that the dose recommended is far below this tolerance. A dose of 100 roentgens may be repeated several times at suitable intervals without danger. The experiments of Peter and of Rohrschneider have shown that roentgen rays can induce cataract when doses beyond the tolerance of the eyelids and conjunctiva are given to the ocular structures, and especially when doses of this magnitude are repeated a number of times. Under these conditions, cataract may develop two, three, or even four years later.

The next question, having to do with chronic inflammations, was actually answered in the paper, though apparently I failed to make my points sufficiently clear. One part of the question appears to assume that roentgen treatment does not have favorable results in chronic inflammations. This is not true. Treatment with roentgen rays often has a favorable result when the lesions are properly treated. Only, in order to produce the most favorable results, larger doses of rays must be employed, and the treatment must be repeated a number of times at suitable intervals. It is well known that, from a pathologic standpoint, chronic inflammations are characterized by more or less leukocytic infiltration, much of which consists of lymphocytes. Besides this, however, there may be varying degrees of proliferation of connective tissue, and of hyaline, caseous, or calcareous degeneration. Under these circumstances, it is not difficult to understand why larger doses are needed and why these doses have to be repeated. In cases of tuberculous adenitis or tenosynovitis, for example, the patient may have to be treated every four weeks for six, eight, or even twelve months. The pathologic process responds favorably in most cases, but this favorable response takes place slowly. In my experience, the best range of dosage in chronic inflammations is from two-thirds to three-fourths of the so-called erythema, or tolerance, dose. For a time which may vary from two to four or five weeks, the discharge from sinuses may increase. After this, however, the discharge gradually diminishes until it finally stops and healing takes place. This is the course usually observed in most tuberculous lesions in which sinuses are present. When sinuses are not present, the lesions usually heal more rapidly; in other words, less treatment is required. In a general way, much the same is true in other chronic inflammations, except that some of them do not have to be treated nearly so long.

The presence of infiltrating leukocytes increases the radiosensitiveness of chronic inflammations. Therefore, the greater the proportion of leukocytic infiltration, the more radiosensitive such lesions tend to be, and *vice versa*. Hyaline, caseous, or calcareous degeneration of cells tends to retard and diminish the effect of the rays. It is obvious that cells undergoing these forms of degeneration are actually dead cells and cannot be influenced by roentgen rays or by any other agent. Another factor which tends to retard or diminish the action of the rays is the presence of proliferating connective tissue. As is well known, this is much less sensitive to roentgen rays than are leukocytes. Consequently, the proportion of proliferating connective tissue or of hyaline, caseous, or calcareous degeneration, on the one hand, and the proportion of leukocytic infiltration, on the other hand, tend to work in opposite directions. The effect of the rays, therefore, may be more or less favorable, depending on the relative proportion of these different factors.

Spontaneous Pneumomediastinum (Mediastinal Emphysema) with Reports of Two Cases in Infants[1]

ARTHUR B. SMITH, M.D.

Assistant, Department of Radiology, Research Hospital, Kansas City, Missouri

and

JOHN F. BOWSER, M.D.

Resident in Radiology, University of Kansas Hospital, Kansas City, Kansas

THE INTERESTING and relatively rare condition of mediastinal emphysema has been described by various authors. Hamman (5, 6) in 1937 published a series of 6 cases and in 1939 reported an additional case. Mediastinal emphysema is preceded in the great majority of instances by interstitial emphysema of the lungs. The air in the interstitial tissues reaches the mediastinum by traveling along connective-tissue bands which surround the bronchi and blood vessels.

The first accurate description of the pathological anatomy of interstitial emphysema of the lungs was by Laennec. The condition had, however, been recognized clinically long before his time by the appearance of air in the subcutaneous tissues about the neck following injury to the chest or forceful distention of the lungs.

Interstitial emphysema often follows trauma to the chest and may occur, also, during induction of therapeutic pneumothorax. A case was reported by Matsuzawa (10) in which an attempt at induction of artificial pneumothorax set up a small tension pneumothorax and allowed the escape of air from the pleural cavity into the mediastinal space and up into the neck. Matsuzawa felt that this was a rather rare complication and attributed it to a tear in the mediastinal pleura. Distention of the lungs produced by forceful muscular effort with the glottis closed may cause interstitial emphysema. It has been observed in new-born infants following attempts at resuscitation and in children after severe paroxysms of coughing. It may also develop during childbirth and after lifting of heavy objects. Spontaneous interstitial emphysema of the lung without a history of trauma or unusual pulmonary distention is not uncommon.

Small amounts of air escaping into the interstitial tissues may give no symptoms or only localized pain. If a larger amount of air escapes, it may form a subpleural bleb or find its way into the mediastinum. In the latter case symptoms may be severe, simulating coronary occlusion. Differential points in the diagnosis include the absence of shock and of myocardial weakness. A distinctive sign of mediastinal emphysema is a peculiar crunching, crackling, bubbling sound heard over the heart with each contraction, due to compression of the air-filled mediastinal tissues lying between the heart and the anterior chest wall. The diagnosis is assured by the appearance of air in the subcutaneous tissues of the neck.

In two of Hamman's (5, 6) seven cases, the interstitial and mediastinal emphysema was accompanied by pneumothorax. This was probably produced by escape of air from the mediastinum. All of the patients recovered with no special treatment other than rest and sedatives.

In an experimental study Macklin (9) was able in some of his animals to demonstrate clearly the point of rupture through the mediastinal wall. Hamman (6), quoting from Macklin's (9) work, states that by direct experiment in animals it was found to be very easy to force air from the mediastinum into the pleural cavity, but it was never possible to force air from the pleural cavity into the mediastinum.

[1] Accepted for publication in October 1941.

Before considering the pathologic anatomy and physiology of pneumomediastinum it may be well to review briefly the normal anatomy of the mediastinum (4). It lies between the right and left pleurae and extends from the sternum in front to the vertebral column behind, and from the upper border of the sternum to the diaphragm. It is divided for purposes of description into an upper and a lower portion. The upper, above the level of the pericardium, known as the superior mediastinum, is bounded below, strictly speaking, by a slightly oblique plane passing backward from the junction of the manubrium and the body of the sternum to the lower part of the body of the fourth thoracic vertebra. Laterally it is bounded by the pleurae, in front by the manubrium sterni, and behind by the upper thoracic vertebrae. The lower portion, below the upper level of the pericardium, is subdivided into three parts: the anterior mediastinum, in front of the pericardium; the middle mediastinum, containing the pericardium and its contents; the posterior mediastinum, behind the pericardium.

The superior mediastinum contains the aortic arch; innominate artery; thoracic portions of left common carotid and left subclavian arteries; innominate veins and upper half of the superior vena cava; the left highest intercostal vein; the vagus, cardiac, phrenic, and left recurrent nerves; the trachea, esophagus, and thoracic duct; the remains of the thymus and some lymph nodes.

The anterior mediastinum contains loose areolar tissues, lymphatic vessels and a few lymph nodes, and the mediastinal branches of the internal mammary artery.

In the middle mediastinum are the heart enclosed in the pericardium; the ascending aorta; the lower part of the superior vena cava with the azygos vein opening into it; the bifurcation of the trachea and the two bronchi; the pulmonary artery, dividing into its two branches; right and left pulmonary veins; the phrenic nerves; some bronchial lymph nodes.

The posterior mediastinum contains the thoracic part of the descending aorta; the azygos and hemiazygos veins; the vagus and splanchnic nerves; the esophagus and thoracic duct; some lymph nodes.

In the demonstration of the pathologic picture of mediastinal and interstitial emphysema, the work of Macklin (8) has played an important part. He has demonstrated the transport of air along the vascular sheaths in experimental animals, after overdistention of the pulmonary alveoli by air. The air finds its way to the vascular sheaths through multiple small ruptures in the alveolar bases overlying the finer ramifications of the pulmonary blood vessels. The pulmonic blood vessels become compressed and arterial and venous circulation is impeded. Similar air-tunneled vascular sheaths have been observed in the human lung and can occur as a result of excessive intrapulmonic pressure during faulty administration of anesthesia by the closed method.

Interstitial emphysema of the lungs is important because of pulmonary circulatory embarrassment and formation of subpleural blebs, which may rupture, causing pneumothorax. Pathologic air transport may distend the mediastinum and further embarrass circulation through pressure on the heart and great vessels, with a fatal outcome. Pneumomediastinum may result in pneumoprecordium, interstitial emphysema of the opposite lung, or pneumoretroperitoneum, and the emphysema may extend to the groin, leg, neck, or head. Such extensions tend to relieve mediastinal circulatory distress.

According to Jessup (7), mediastinal emphysema may occur as a complication in a great variety of conditions. It has been known to result from injuries and diseases of the lung, from perforations of the chest wall, artificial pneumothorax, perforating wounds of the larynx, trachea, and bronchi; wounds of the neck and diaphragm; also from rupture of the esophagus. In one case a rupture of the stomach produced retroperitoneal emphysema which extended to the mediastinum.

Mechanisms of production are numer-

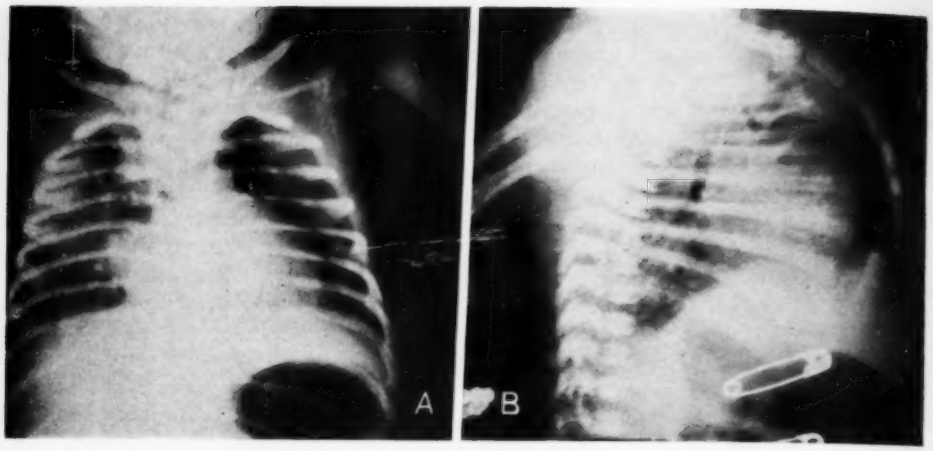


Fig. 1. Case 1: A. Roentgenogram 36 hours after birth, showing air in the anterior mediastinum and between the layers of the cervical fascia on both sides. B. Lateral view, showing air in the anterior and superior mediastinum; the heart is compressed and displaced posteriorly.

ous, but symptoms are constant and significant.

The effects seem to be the result of increased mediastinal pressure. The outstanding effect is a mechanical interference with circulation brought about by compression of the large veins entering both sides of the heart. Most of the outward signs observed are dependent on this blockage of circulation. Symptoms and physical signs most commonly present are extreme cyanosis and dyspnea, rapid pulse, congestion of the superficial veins of the neck, and low blood pressure. Subcutaneous emphysema is usually present.

Many cases end fatally, but death may be avoided in some instances by early recognition of the condition and the application of suitable treatment. The latter Jessup considers from two aspects: controlling the source of air and giving an outlet to the air already accumulated.

Fisher and Macklin (2) described a fatal case of pulmonic interstitial emphysema occurring in a child of 22 months, arising from aspirated pieces of peanut. The pathologic picture was that of a pulmonic, perivascular emphysema of the left lung and in the hilus region of both right and left lungs, together with the presence of large bubbles of air in the anterior and superior mediastinum and in the loose tissue overlying the parietal pericardium. This caused compression of, and obstruction to, the pulmonary and mediastinal circulation, producing severe dyspnea, cyanosis, and anoxemia, as well as impeded action of the heart. Circulatory embarrassment from the pressure of this emphysematous air was the principal cause of death.

A case of mediastinal emphysema associated with a spontaneous pneumothorax on the left was presented by Morey and Sosman (12). The patient was a young adult who had had a spontaneous pneumothorax on the right side sixteen months previously. On auscultation over the chest a peculiar knock or click, resembling at times the squeak of a leather saddle, was clearly heard. The patient made a complete recovery with no treatment other than rest and sedatives. The crackle in the chest persisted for two weeks and the pneumothorax had completely disappeared in three weeks.

The seriousness of this condition depends upon the amount of air that escapes from the lungs and the pressure that is built up in the mediastinum. Ballon and Francis (1) have shown that the blood vessels of the mediastinum are the first

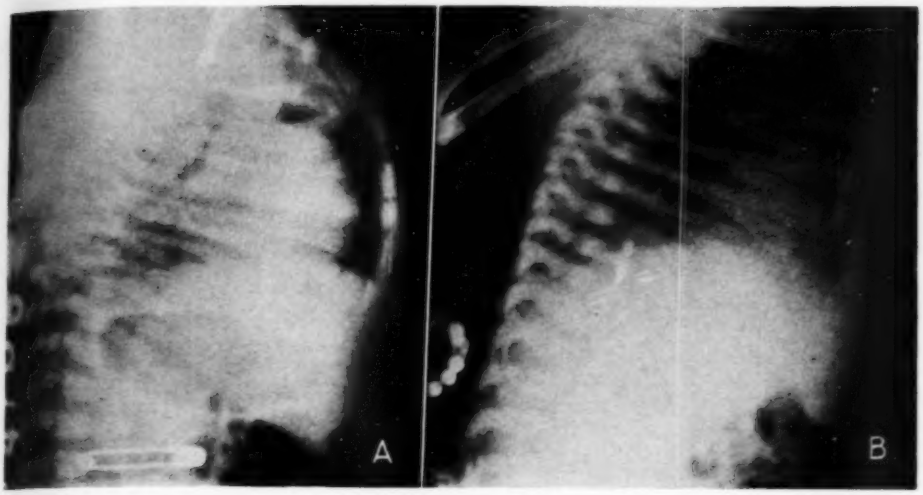


Fig. 2. Case 1: A. Roentgenogram made immediately after withdrawal of air, showing much less air in the mediastinum and less displacement of the heart. B. One week later there is essentially complete absorption of the mediastinal air.

structures to be compressed. This in turn produces cyanosis and air hunger. Cutting the skin over the neck to allow air to escape has been done.

Graebner (3) reported three cases where pneumomediastinum occurred in patients with acute obstructive laryngitis. The characteristic crackling murmur over the precordium was noted. The extrapulmonary air was quickly absorbed after its continued extravasation was stopped by elimination of the obstruction. Graebner states that the occurrence of pneumomediastinum is directly influenced by obstruction of the respiratory tract and "is not directly due to nor proximately the result of tracheotomy *per se*."

In contrast to the above, Michels (11) presented six cases of acute laryngeal obstruction, in five of which pneumothorax and mediastinal emphysema followed tracheotomy. Three of the six patients died. Two recovered with no primary treatment of the pneumothorax and one with continuous closed drainage.

Stenbuck (13) believes that diffuse subcutaneous emphysema in traumatic cases usually arises superficially in the chest wall and not in the mediastinum. The commonest causes on a traumatic service are penetrating wounds of the chest and fractured ribs causing laceration of the lung. Only two cases of primary mediastinal emphysema were seen by this writer, both in patients with severe crushing injuries of the entire chest.

CASE REPORTS

CASE 1: Baby C., a premature male infant (7 months gestation), was delivered spontaneously at 7:00 A.M. Dec. 11, 1940. The baby was quite cyanotic; respirations were shallow. Carbon dioxide and oxygen were administered but there was no improvement. The temperature was 99° F. at the end of the first twenty-four hours, and thereafter varied from 98° to 98.6°. The white blood count was 20,650 at the end of forty-eight hours, with 83 per cent polymorphonuclears.

Thirty-six hours after birth anteroposterior (Fig. 1-A) and lateral (Fig. 1-B) negatives of the chest were made and air was seen in the anterior and superior mediastinum and between the layers of the cervical fascia on both sides. A needle was introduced under the sternum in the fourth anterior interspace on the right and the escaping air forced the plunger out of the syringe attached to the needle. A negative made immediately following this (Fig. 2-A) revealed much less air in the mediastinum, and a week later (Fig. 2-B) all of the mediastinal air had been absorbed.

CASE 2: Baby S., a premature female infant (8 months gestation), was delivered spontaneously

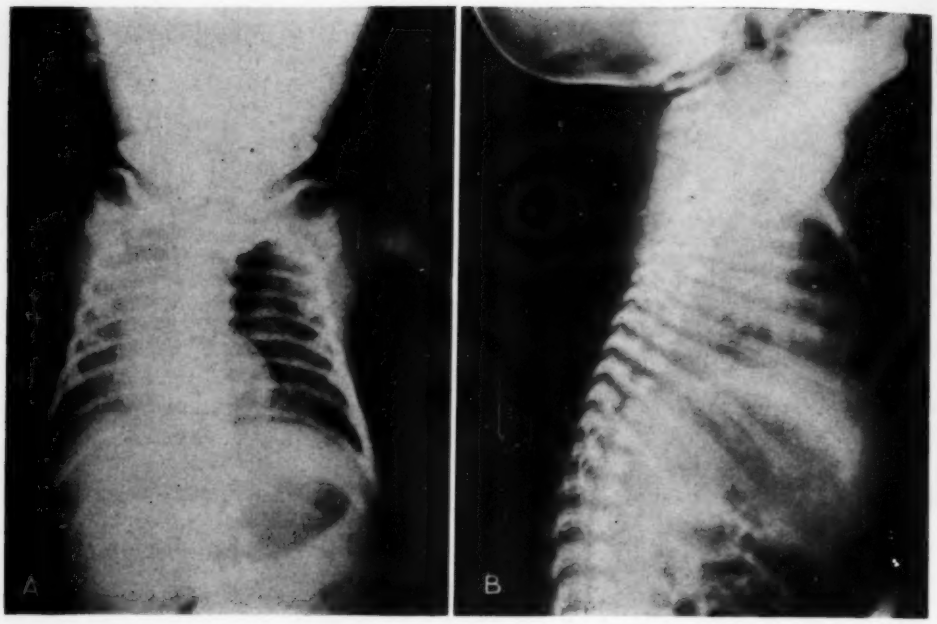


Fig. 3. Case 2: A. Roentgenogram made 72 hours after birth, showing air in the anterior mediastinum. B. The lateral negative shows air in the superior and anterior mediastinum with slight posterior displacement of the heart.

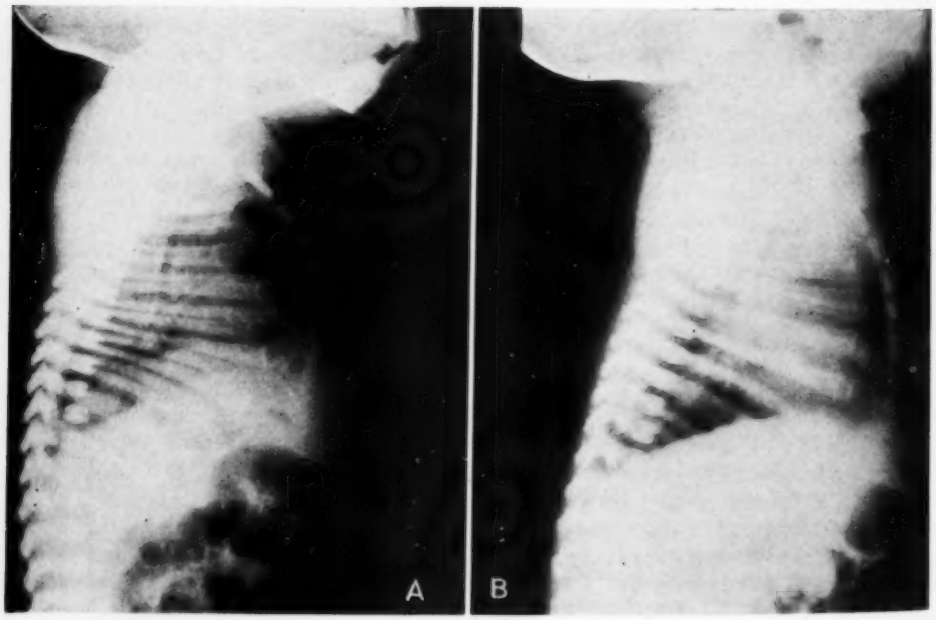


Fig. 4. Case 2: A. Three days after the roentgenograms shown in Fig. 3 were made the air in the mediastinum is seen to be decreased in amount. B. One month after birth there is no evidence of mediastinal air.

of a primiparous mother following labor of four hours' duration on Feb. 2, 1941. The baby was cyanotic at birth and remained so for two days. There was no abnormality in the size, shape, or respiratory movements of the chest. Upon crying the baby became a normal red color. Physical examination of the heart was negative. Breath sounds could be heard only over the hilar portions of the lungs. The temperature was 97° at birth and in the hospital ranged between 97° and 99°.

Twenty-four hours following birth a routine anteroposterior negative of the chest was made which was interpreted as showing left pneumothorax with right congenital atelectasis. Forty-eight hours later anteroposterior (Fig. 3-A) and lateral negatives (Fig. 3-B) of the chest were made which showed air in the anterior and superior mediastinum and no pneumothorax. A lateral negative of the chest (Fig. 4-A) was made on the sixth day of life and the air shadow in the anterior mediastinum was present but smaller in amount.

No attempt at removal of the mediastinal air was made. The cyanosis disappeared on the third day of life and the patient had an uneventful hospital course.

On March 3, 1941, an opportunity was afforded to re-examine the chest because of an upper respiratory infection. Roentgenograms in the anteroposterior and lateral (Fig. 4-B) projections failed at that time to show any mediastinal air.

SUMMARY

The literature on mediastinal emphysema and interstitial emphysema of the lungs has been reviewed and two additional cases are presented. Pathologic air transport along vascular sheaths has been definitely demonstrated, both in experimental animals and in man. The condition is probably more common than has heretofore been realized and should be considered in the differential diagnosis in all cases of chest injury and in cases of precordial pain without the usual findings of coronary occlusion. In infants particularly, the combination of persistent dyspnea with signs of increasing circulatory embarrassment warrants consideration of the possibility of pneumomediastinum, as relief of increased mediastinal pressure may be life-saving. Roentgenography of the chest, especially in the lateral projection, is diagnostic. Air between the layers of the cervical fascia may be apparent on the negative before it is possible to detect it clinically.

Research Hospital, Kansas City, Missouri

BIBLIOGRAPHY

1. BALLON, H. C., AND FRANCIS, B. F.: Consequences of Variations in Mediastinal Pressure: Mediastinal and Subcutaneous Emphysema. Arch. Surg. **19:** 1627–1659, December 1929. Quoted by Morey and Sosman (12).

2. FISHER, J. H., AND MACKLIN, C. C.: Pulmonic Interstitial and Mediastinal Emphysema: Report of a Fatal Case in Which the Emphysema Occurred in a Child as a Result of Aspiration of Peanut Fragments. Am. J. Dis. Child. **60:** 102–115, July 1940.

3. GRAEBNER, H.: Pneumopericardium and Pneumomediastinum in Cases of Acute Obstructive Laryngitis. Arch. Otolaryng. **29:** 446–456, March 1939.

4. Gray's Anatomy. Philadelphia, Lea & Febiger 1936.

5. HAMMAN, LOUIS: Spontaneous Interstitial Emphysema of the Lungs. Tr. A. Am. Physicians **52:** 311–319, 1937.

6. HAMMAN, LOUIS: Spontaneous Mediastinal Emphysema. Bull. Johns Hopkins Hosp. **64:** 1–21, January 1939.

7. JESSUP, P. M.: Mediastinal Emphysema. Arch. Surg. **23:** 760–782, November 1931.

8. MACKLIN, C. C.: Transport of Air along Sheaths of Pulmonic Blood Vessels from Alveoli to Mediastinum: Clinical Implications. Arch. Int. Med. **64:** 913–926, November 1939 (as edited in the Yearbook of General Medicine, 1940).

9. MACKLIN, C. C.: Pneumothorax with Massive Collapse from Experimental Local Over-Inflation of the Lung Substance. Canad. M. A. J. **36:** 414–420, April 1937. Quoted by Hamman (6).

10. MATSUZAWA, D.: Mediastinal Emphysema as a Complication of Artificial Pneumothorax: Report of Case. Quart. Bull. Sea View Hosp. **2:** 173–178, January 1937.

11. MICHELS, M. W.: Pneumothorax and Mediastinal Emphysema Complicating Tracheotomy. Arch. Otolaryng. **29:** 842–852, May 1939.

12. MOREY, J. B., AND SOSMAN, M. C.: Spontaneous Mediastinal Emphysema, with Report of Case Associated with Spontaneous Pneumothorax. Radiology **32:** 19–22, January 1939.

13. STENBUCK, J. B.: Traumatic Subcutaneous Emphysema of Thoracic Origin. N. Y. State J. Med. **37:** 395–399, Feb. 15, 1937.

Roentgen Manifestations of Bejel ("Endemic Syphilis") as Observed in the Euphrates River Valley[1]

GLENN S. ROST, M.D.

Deir-ez-Zor, Syria

BEJEL IS A treponematosis endemic among the nomad and semi-nomad inhabitants of the Euphrates River Valley. Its clinical description has initiated a reconsideration of certain differences between syphilis and yaws and has led to a consideration of the relationship of bejel to these disease entities.

Inasmuch as the roentgenologic manifestations of bejel provide another approach to its study, it is proposed to set forth briefly the major diagnostic features of the disease demonstrable by roentgen examination.

E. H. Hudson first accurately described bejel, and others have already portrayed the clinical manifestations of this childhood treponematosis. These writers have called attention to its predilection for skin, mucous membrane, and osseous system, and also to the absence of involvement of the cardiovascular and central nervous systems. Hudson (7), referring to the bone changes, says of the disease: "Periostitis and bone necrosis are found in both early and late lesions." Elsewhere (5) he states that it is "not productive of apparent lesions in the nervous, cardiovascular, or other interstitial organs . . . ."

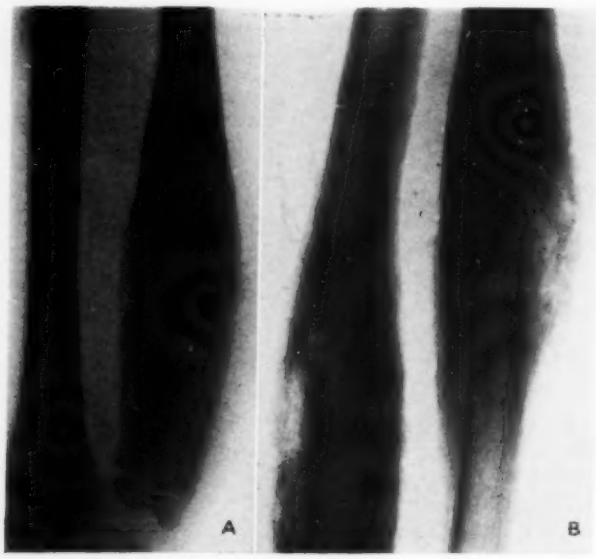


Fig. 1. A. Bejel in the ulna of a ten-year-old child. Slight swelling was noted for one and one-half years. Rapid increase in size during the past two months.

B. Advanced periosteal thickening as the result of bejel contracted in childhood. Symptoms for six months only. Note the moth-eaten appearance and the change in character of the periosteal proliferation.

The bone lesions of bejel have many features in common with those of acquired syphilis and to some extent also with those

[1] From the Deir-ez-Zor Medical Center. Accepted for publication in December 1941.

seen in late heredosyphilis. Lesions similar to the early lesions of heredosyphilis have not been observed in our studies thus far.

Bejel of the bones is predominantly a periosteal and endosteal proliferation with varying degrees of rarefaction. The shafts of the long bones are most frequently involved, especially the tibia and ulna, although no bone is exempt. These lesions have not been observed below the age of five years.

The early manifestations are primarily those of a periosteal thickening with resultant increase in bone caliber. The periosteal new bone is most frequently seen in layers parallel to the long axis of the bone. The result is usually a dense, irregular or spindle-shaped thickening of the cortical bone with variable but usually minimal changes in the medullary space. This may be associated with increased density of the underlying bone at later stages. The distal third of the ulna of the ten-year-old boy seen in Figure 1-A shows the lesion. This patient's blood Kahn reaction was four plus. Rarefaction, when it occurs in these lesions, leaves a rather characteristic moth-eaten appearance of the periosteal new bone.

As time passes, the periosteal surface may take on a fuzzy appearance, amounting to the development of trabeculae of new bone lying at right angles to the shaft, as in osteosarcoma. A picture very similar to the so-called "lace work" pattern of acquired syphilis may also develop. Changes of this type may be observed in Figure 1-B.

These are progressive steps toward a type of lesion in which the new bone completely fills the medullary cavity. The areas of decreased density extend into the medullary cavity, the composite picture being one of an irregularly thickened shaft made up of periosteal and endosteal new bone with irregular areas of rarefaction outlined against a background of increased density.

The bone lesion itself may be either diffuse or localized. When diffuse, the lesion is essentially one of sclerosis of the cortex. Its distribution may be patchy. Associated with the increased density there is usually an increase in caliber of the bone. Areas of rarefaction may appear either in the cortex or centrally. When central, these areas, not unlike the gummata of late syphilis, may be accompanied by a peripheral zone of subperiosteal new bone formation and condensation, causing a localized thickening of the shaft. This involvement of the shaft may extend to the end of the bone and reach the joint. The joint involvement is most often localized, as in acquired syphilis, to one articular surface. This phase of involvement will be dealt with more fully presently. In the late stages of the active diffuse lesion, involvement may be extensive, completely obliterating normal bone configuration and resulting in a concentric enlargement of the bone, irregular in contour and with uniformly increased density.

Fig. 2. A. Forearm of a six-year-old child who had bejel one year previously. All stages of periosteal and endosteal involvement are shown.
B. Roentgenogram of the leg made at the same time as that of the forearm.

The patient represented in Figures 2-A

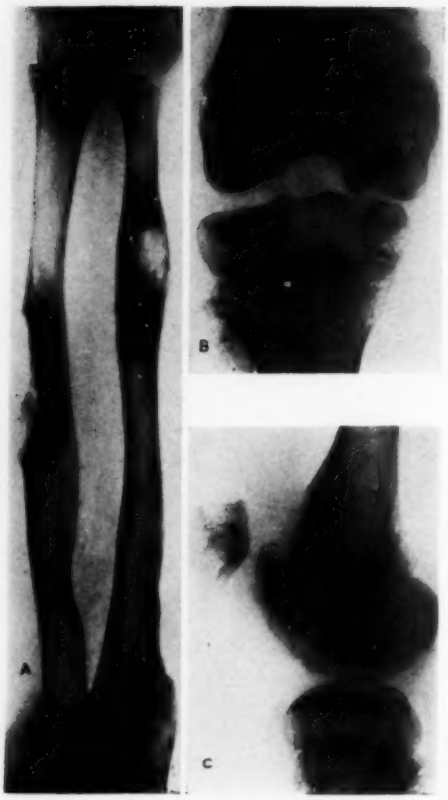


Fig. 3. A. Roentgenogram made five months after the onset of nocturnal bone pain and deformity, and three years after initial infection. Involvement of the elbow joint and of the epiphysis of the radius, and dactylitis of the first metacarpal bone are demonstrable on the original film.
B and C. The knee joint of the same patient. Erosion of one articular surface, as well as epiphyseal and metaphyseal involvement, is present. This case demonstrates the difficulty of differentiation from tuberculosis.

and 2-B had clinical bejel with an associated rash at the age of five, one year before the roentgenograms were made. Here are demonstrated practically all phases of involvement, including both early and advanced periosteal proliferation, endosteal changes and areas of destruction not unlike the gummata of late syphilis.

At times one sees large areas of destruction of the cortex with variable reduction in density and minimal new bone formation. The roentgenograms reproduced in Figure 3 are of a twelve-year-old boy who had clinical bejel three years before he presented himself for treatment. Nocturnal bone pain and deformity of five months' duration finally caused him to seek medical aid. His blood Kahn reaction was three plus. Involvement of one

Fig. 4. Sequestration is rarely seen. This film is presented through the courtesy of E. H. Hudson.

articular surface each in the knee and in the elbow is worthy of note.

Another phase of the disease is represented by localized gummata-like lesions, seen most often in the anterior surface of the tibia, and usually associated with a marked degree of increased cortical density and thickening, as shown in Figure 5-A. Here again bone destruction and bone proliferation are seen in an ever-changing ratio. In the untreated lesion these areas

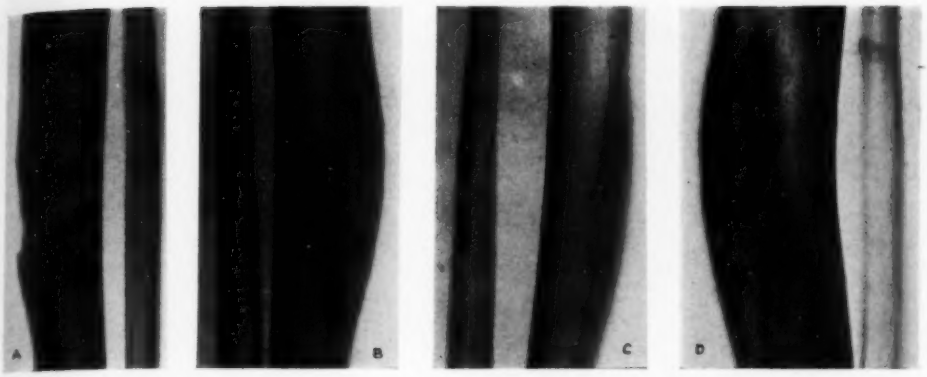


Fig. 5. A. Localized lesion in a boy fourteen years of age. The soft palate had been destroyed by a typical ulcer of late bejel.
B. Untreated localized involvement in another boy fourteen years of age.
C. Same case as in B, one month after induction of treatment. The patient received 0.75 gm. of metal during the interval.
D. The tibia as seen in bejel of twelve years' standing. The patient was thirty-five years old and had had no treatment. There was no complaint referable to this region. Note the variation in contour and density.

of increased density and proliferation are usually accompanied by at least one large or several smaller well demarcated areas of destruction extending deeply into cortical bone. Sequestration (Fig. 4) may occur in such lesions but is not the rule.

Healing may occur spontaneously, leaving only a certain degree of local irregularity in contour and density, or a uniform irregularity similar in all points to the saber tibia with its variation in contour, density, and bone pattern, and with loss of the medullary cavity as such (Fig. 5-D).

Each of these lesions responds readily to small amounts of anti-syphilitic medication, less than a gram of bismuth metal rendering any one of them temporarily asymptomatic and producing x-ray evidences of the healing process. The most notable changes occurring early in treatment are regression of the periosteal changes, deposition of radio-visible bone in the existing defects, and decrease of cortical density. Figures 5-B and 5-C portray the effect obtained in one month by the use of 0.75 gm. of bismuth metal.

Roentgen-ray and fluoroscopic evidences of cardiovascular involvement have not been encountered under circumstances irrefutably attributable to bejel. Likewise articular manifestations comparable to Charcot's joints have not been seen. In these points, bejel seems to differ from syphilis, and the difference may prove to be important in differentiation.

Joint involvement other than the neuropathic type, while not common, is encountered. These lesions closely resemble those of the syphilitic infections. The site of involvement may be the joint surface itself, the epiphysis, or even the metaphysis; diffuse bone involvement is often co-existent. Destruction of one articular surface with irregular erosions in the underlying epiphysis but without involvement of the contiguous joint surface is the type of lesion most frequently seen. Here, as elsewhere, there is a variable amount of proliferative change surrounding the area of lysis (Fig. 3). F. Campbell Golding (2) has said of syphilitic joint involvement: "The presence of multiple irregular areas of an osteoplastic type in the shaft with discrete areas of subchondral erosion and the absence of general osteo-porosis and haziness of bone should serve to differentiate the disease from tuberculosis." This statement applies equally well to bejel.

Characteristically the subjects of the

Fig. 6. Destruction of the maxilla. The soft tissue prominence is the upper lip. The nose is faintly outlined above it. Following diagnostic aspiration and collapse of the lesion this patient was refused passage by plague quarantine officials on the grounds that she was not the same patient that had been passing daily for treatment.

present study all gave a history of bejel in infancy or at periods ranging from one to thirty-nine years prior to the time of the x-ray examination. This coincides with the experience of Hudson and Crosley (7), who in a series of 1,000 analyzed cases obtained a history of infection occurring in childhood in over 76 per cent. Practically without exception the blood serology in our group was positive. Commonly accepted clinical criteria of heredosyphilis were absent.

COMMENT

Holmes and Lingley (4), speaking of acquired bone syphilis have said: "The combination of bone destruction with irregular and usually marked bone production in the same lesion should always make one suspicious of syphilis." The statement is no less true of bejel.

Pendergrass, McLean, Hodges, and others have each, in their descriptions of syphilis of the osseous system, portrayed pictures which might well have been those of certain phases of bejel. There are, however, notable exceptions. Thus, evidence of the counterpart of osteochondritis as seen in early heredosyphilis is lacking in bejel, and the entire absence of neuropathic joint manifestations may also be mentioned. Roentgen evidences of cardiovascular involvement have not been found.

These features are of major importance in the attempt to correlate syphilis, bejel, and yaws. In these respects the x-ray findings substantiate further certain similarities and differences, already recognized clinically. The inherent relationship between these respective infections cannot be ignored. But it also becomes imperative that any premature attempt to place bejel unreservedly under the heading of syphilis must be held in abeyance. The final classification must wait until such time as studies on the epidemiology, of modes of transmission, and of clinical variances, clarify the entire picture sufficiently to prove or disprove such a relationship.

SUMMARY

A brief description of the roentgen findings of bejel is presented.

The osseous changes of bejel consist essentially of periosteal and endosteal proliferation with varying degrees of rarefaction resembling at times the gummata of late syphilis.

The clinical differences and similarities between syphilis and bejel are further substantiated by the roentgen manifestations.

Attention is directed to the absence of roentgen ray evidences similar to those seen in early heredosyphilis.

The absence of neuropathic joint lesions is noted.

It is suggested that final classification of the disease in its relationship to syphilis and yaws be reserved pending the nar-

rowing of the present hiatus in our knowledge of these disease entities.

Appreciation is expressed to A. Oppenheimer for his assistance and advice in the preparation of this manuscript.

Hertzler Clinic, Halstead, Kansas

BIBLIOGRAPHY

1. CAFFEY, J.: Syphilis of Skeleton in Early Infancy; Non-Specificity of Many of the Roentgenographic Changes. Am. J. Roentgenol. **42:** 637–655, November 1939.

2. GOLDING, F. C.: in A Textbook of X-Ray Diagnosis, edited by Shanks, S. C., Kerley, P., and Twining, E. W., London, H. K. Lewis and Co., 1939.

3. HODGES, P. C.: in Diagnostic Roentgenology, edited by Golden, R., New York, Thomas Nelson & Sons, 1938, pp. 387–398.

4. HOLMES, G. W., AND LINGLEY, J. R.: Roentgenologic Manifestations of Syphilis. New England J. Med. **217:** 983–987, Dec. 16, 1937.

5. HUDSON, E. H.: Mucocutaneous Syphilis (Bejel) in Syria: Results of Dark-Field Examination. New England J. Med. **215:** 392–396, Aug. 27, 1936.

6. HUDSON, E. H.: Significance of Bejel. Publications, American Association for the Advancement of Science, No. 6, 1938, pp. 35–39.

7. HUDSON, E. H., AND CROSLEY, S. S.: Bismuth in Treatment of Endemic Syphilis. J. Trop. Med. **39:** 245–250, Nov. 2, 1936.

8. MACQUEEN, J.: Syphilis Insontium in Palestine. Brit. J. Ven. Dis. **10:** 33–50, January 1934.

9. MCLEAN, S.: Roentgenographic and Pathologic Aspects of Congenital Osseous Syphilis. Am. J. Dis. Child. **41:** 130–152, January 1931.

10. MCLEAN, S.: Correlation of Roentgenographic and Pathologic Aspects of Congenital Osseous Syphilis, Am. J. Dis. Child. **41:** 363–395, February 1931.

11. PENDERGRASS, E. P., GILMAN, R. L., AND CASTLETON, K. B.: Bone Lesions in Tardive Heredosyphilis. Am. J. Roentgenol. **24:** 234–257, September 1930.

12. PENDERGRASS, E. P., AND BROMER, R. S.: Congenital Bone Syphilis; Preliminary Report; Roentgenologic Study with Notes on Histology and Pathology of Condition. Am. J. Roentgenol. **22:** 1–21, July 1929.

13. STOKES, J. H.: Modern Clinical Syphilis, Philadelphia, W. B. Saunders and Co., 1934.

Congenital Atresia of the Esophagus[1]

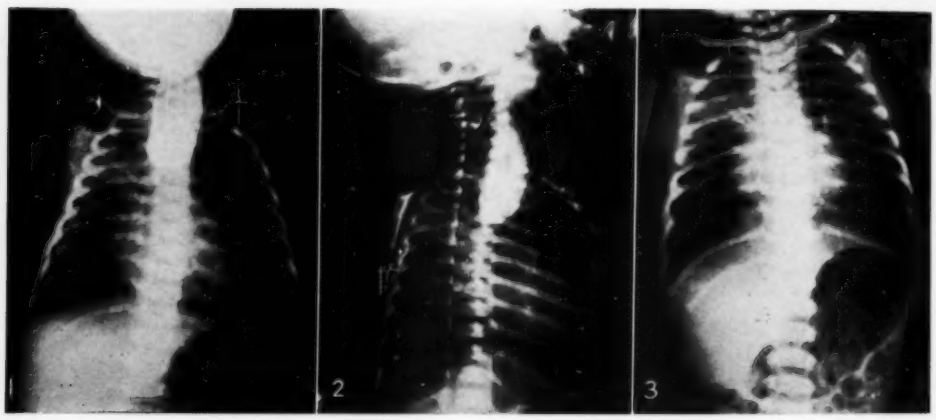
MURRAY FUHRMAN, M.D., DAVID DRAGUTSKY, M.D., GEORGE ROOK, M.D., and LEO GROSSMAN, M.D.

Metropolitan Hospital,[2] New York, N. Y.

CONGENITAL atresia of the esophagus has usually been regarded as an uncommon condition. In 1931, Rosenthal (10) recorded 255 cases from the literature, including a number of his own. Since then numerous additional cases have been published. We recently encountered two examples, associated with tracheo-esophageal fistula, within a month. During the previous ten years there had been 12,285 deliveries on the maternity service of Metropolitan Hospital, but among these the records fail to show a single case of esophageal atresia. Nor is it likely that any cases were overlooked, as a very high percentage of autopsies on the newborn is obtained at this institution, particularly in cases with unusual symptoms.

The purpose of the present communication is to present our cases, to review briefly the salient clinical and radiographic features, and to describe a new and simple diagnostic procedure—aero-esophagography—for establishing a diagnosis of congenital atresia of the esophagus.

A number of excellent reviews have appeared in the American and English literature, notably those of Griffith and Lavenson (3) in 1909, Cautley (2) in 1917, Plass (9) in 1919, Vogt (14) in 1929, Rosenthal (10) in 1931, Mathieu and Goldsmith (8) in 1933, and Lanman (5) in 1940.



Figs. 1–3. Case 1: The upper esophageal segment is visualized terminating in a dilated, blind pouch. The stomach is distended with gas. The trachea and bronchi (Fig. 3) are outlined by the contrast medium. Some of the barium formula has apparently been aspirated.

Vogt classifies congenital atresia of the esophagus into the following categories:

Type 1: Complete absence of the esophagus (extremely rare).

Type 2: Complete atresia with no tracheo-esophageal fistula (rare).

Type 3A: The upper esophageal segment communicates with the trachea (rare).

Type 3B: The upper segment terminates blindly; the lower communicates with the trachea or with the bronchi near the level of bifurcation of the trachea (the commonest type).

[1] Accepted for publication in October 1941.

[2] The Department of Radiology and the Department of Pediatrics.

Type 3C: Both esophageal segments communicate with the trachea or bronchi (rare).

Our cases are both of Type 3B (Type 3 of Ballantyne, 1).

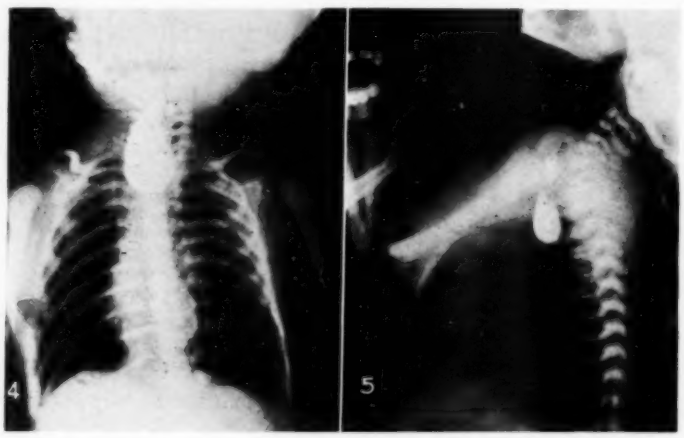
CASE REPORTS

CASE 1: R. K., a white male, was born Oct. 1, 1940, after a normal pregnancy and labor. The family history is irrelevant. The infant appeared normal at birth but shortly thereafter drooling and the presence of a large amount of thick, frothy mucus in the mouth and pharynx were observed, and repeated aspiration became necessary to keep the pharynx clear. On the following day the temperature rose to 101°, the respiratory rate increased, and many medium and coarse râles were heard throughout the chest. A roentgenogram showed consolidation of the upper lobe of the right lung. Regurgitation of all feedings occurred, and several attempts at gavage proved unsuccessful. It appeared, however, as if some of the food was retained when a Breck feeder was used. The pneumonia was considered the sole cause of coughing, respiratory difficulty, and vomiting. Clyses and whole blood transfusions helped to maintain fair hydration, adding to this impression. The persistence of symptoms, however, led to the consideration of esophageal atresia as the underlying condition, and a roentgen investigation was requested.

On Oct. 5 a soft rubber catheter was introduced into the esophagus, and under fluoroscopic guidance was passed down toward the stomach. An obstruction was met with at the level of the third dorsal vertebra. Efforts to pass the catheter beyond this point caused the tip to coil up.

A thin barium preparation was then given—inadvisedly—and roentgenograms were taken (Figs. 1 and 2). The upper esophagus appeared as a dilated smooth pouch. Gaseous distention of the stomach and small intestines was present. Shortly after the exposures were made, the infant gagged and regurgitated some of the barium mixture. A subsequent film (Fig. 3) showed some of the contrast medium in the bronchial tree—aspiration of the barium formula having apparently occurred.

A gastrostomy was performed on the same day. On the following day the temperature rose to 106°, and there was considerable respiratory embarrassment. The child became markedly cyanotic, and thick, gray, blood-streaked, frothy mucus was repeatedly aspirated from the pharnyx. Death occurred that evening.



Figs. 4 and 5. Case 2: The upper esophageal segment is outlined by the instilled iodized oil.

At autopsy the upper third of the esophagus was found to terminate in a blind pouch at the level of the third thoracic vertebra. The lower segment of the esophagus commenced immediately below, was somewhat narrowed above, but regained its normal caliber as it descended toward the stomach. A narrow fistulous communication existed between the proximal end of the lower segment and the trachea, 1 cm. above its bifurcation. Patchy areas of consolidation were present in both lungs.

Anatomical Diagnosis: Atresia of the esophagus with tracheo-esophageal fistula; confluent bilateral bronchopneumonia.

CASE 2. R. P., white female, was born on Oct. 27, 1940. Forceps delivery was necessary, following a long arduous labor, and resuscitation was difficult. Marked cyanosis was present, much thick mucus being aspirated from the pharynx. The family history was irrelevant. Physical examination shortly after birth was negative, apart from some coarse moist râles throughout both lung fields.

On the following morning (Oct. 28) the cyanosis

persisted, and inspiratory stridor with suprasternal retraction was noted. At this time the possibility of cerebral hemorrhage or laryngeal obstruction was considered. During the day the infant regurgitated every feeding after about one-half ounce had been taken, and there was an increase of the coughing, dyspnea, and cyanosis at these times. These findings suggested the presence of an esophageal atresia, and the infant was referred for roentgen studies.

A catheter was introduced into the esophagus, but could not be passed beyond the level of the bifurcation of the trachea. A few cubic centimeters of iodized oil were then passed through the catheter into the esophagus. The oil collected in a blind cul-de-sac. Roentgenograms were then taken (Figs. 4 and 5), confirming the fluoroscopic findings. Distention of the stomach and intestinal tract with gas was noted. Following the roentgen examination, the oil was removed from the esophageal pouch by suction.

Gastrostomy was performed on Oct. 27. The child did well for a fortnight, receiving breast milk *via* the gastrostomy tube and supportive therapy. The tube became loose on Nov. 9 and again on Nov. 15 and had to be replaced. Death occurred on Nov. 16.

At autopsy the upper portion of the esophagus was found to end blindly in a dilated sac about 5 cm. below the pharynx. The lower segment of the esophagus was separated from the upper sac by a small amount of connective tissue. A fistulous channel connected the lower segment with the trachea, immediately above the bifurcation. The lungs were essentially negative. The abdomen showed signs of peritonitis. An infected fistulous tract ran from the gastrostomy wound to the stomach. A separate communication with the peritoneal cavity could not be demonstrated.

Anatomical Diagnosis: Atresia of the esophagus with tracheo-esophageal fistula; generalized peritonitis.

Though a successful surgical intervention for congenital atresia of the esophagus has not as yet been reported, we feel that the future should bring accounts of such cases. It behooves the pediatrician to be on the lookout for suggestive clinical symptomatology, and to refer the infant for prompt roentgen investigation when the suspicion of esophageal atresia exists. Constant drooling, the reappearance of frothy mucus in the mouth and pharynx following frequent pharyngeal aspiration, repeated regurgitation after feeding, followed by coughing, cyanosis, and dyspnea, are so characteristic of congenital atresia of the esophagus that if the condition is kept in mind, a strong presumptive diagnosis can readily be made at an early hour. Sometimes, however, the clinical picture may be confusing, and the preponderance of pulmonary symptomatology may cause one to overlook the underlying pathology, as in the first of our two cases. Roentgen studies will establish the diagnosis.

The roentgenologist should use no procedure in his investigations which will lessen the chances of survival of the infant or interfere with subsequent surgical intervention. The use of a formula containing barium as a contrast medium for the demonstration of esophageal atresia is definitely contraindicated. The aspiration of an appreciable amount of this mixture will usually lead to a fatal bronchopneumonia. If a liquid contrast medium is employed, it should be a bland iodized oil or aqueous solution. Though the aspiration of such an oil is regarded as relatively harmless, we feel that it is not completely so, particularly in this condition, where aspiration pneumonia may have already taken place. If oil is used, it should be removed from the esophagus by suction immediately after the roentgen studies have been completed.

The diagnosis of congenital atresia of the esophagus can, however, be established without the use of liquid contrast media. Tucker and Pendergrass (13) used a 3.5 mm. esophagoscope and a 3.5 mm. bronchoscope for direct visualization of the esophageal pouch and the tracheo-esophageal fistula, respectively. They passed a small opaque ureteral catheter through the bronchoscope, into the fistulous opening and on into the lower esophageal segment, demonstrating its passage into the stomach fluoroscopically. Solis-Cohen and Levine (11) described a "pencil-like" airway leading from the bifurcation of the trachea to the stomach; this they believed represented the lower segment of the esophagus filled with air. They point out that air cannot be demonstrated in the upper esophagus, which is filled with retained secretions. These diagnostic criteria, however,

we have found unreliable. Normal bronchovascular markings in the lower lung fields in the lateral position may form a configuration which closely simulates a "pencil-like" airway. Furthermore, the lower esophagus may be clearly demonstrated in a normal infant (in the absence of a tracheo-esophageal fistula) as the result of regurgitation of gas from the stomach. The absence of air in the upper esophagus is also an untrustworthy sign. Its presence frequently cannot be demonstrated in normal infants. We also see no reason why air cannot be occasionally demonstrated in the esophageal pouch of a case of congenital atresia following regurgitation of the contents of the sac.

We would suggest the following simple, harmless procedure as a means of establishing the diagnosis of congenital atresia of the esophagus without the use of a liquid contrast medium. A soft rubber catheter is introduced through a naris into the esophagus, and under fluoroscopic visualization is passed downward toward the stomach. It will not pass beyond the point of atresia. With the patient in the Trendelenburg position, a small bulb syringe is attached to the proximal end of the catheter, and a few cubic centimeters of air are injected. The air will clearly outline the esophageal pouch. The presence of gas in the gastro-intestinal tract indicates the presence of a fistulous communication between the lower segment of the esophagus and the respiratory passages (trachea or bronchus). Films in the Trendelenburg position may then be taken to illustrate the fluoroscopic findings. This method of aeroesophagography renders the use of a liquid contrast medium unnecessary.

SUMMARY

1. Two cases of congenital atresia of the esophagus with tracheo-esophageal fistula are presented.

2. The characteristic clinical features and the importance of an early diagnosis are considered.

3. The use of liquid contrast media for visualization of the esophagus and possible harmful sequelae are discussed.

4. A new, simple, harmless procedure—aeroesophagography—is proposed, whereby the upper esophageal segment may be demonstrated by the injection of air through a soft rubber catheter introduced into the esophagus.

We extend our thanks to Dr. Reuel A. Benson, Director of the Department of Pediatrics, and Dr. Tobias B. Weinberg, Director of the Department of Radiology, for their co-operation and helpful suggestions.

Metropolitan Hospital, Welfare Island, N. Y.

BIBLIOGRAPHY

1. BALLANTYNE, J. W.: Manual of Antenatal Pathology and Hygiene: The Embryo. Edinburgh, Wm. Green & Sons, 1904, p. 462.

2. CAUTLEY, E.: Malformation of the Œsophagus. Brit. J. Child. Dis. **14:** 1–10, 1917.

3. GRIFFITH, J. P. C., AND LAVENSON, R. L.: Congenital Malformation of the Esophagus, with Report of a Case. Arch. Pediat. **26:** 161–169, 1909.

4. HEATLY, C. A.: Some Problems in Esophageal Atresia. Ann. Otol., Rhin. & Laryng. **45:** 1122–1132, 1936.

5. LANMAN, T. H.: Congenital Atresia of Esophagus; Study of 32 Cases. Arch. Surg. **41:** 1060–1083, 1940.

6. LEVEN, N. L.: Surgical Management of Congenital Atresia of Esophagus with Tracheo-esophageal Fistula: Report of 2 Cases. J. Thoracic Surg. **6:** 30–39, 1936.

7. MANGES, W. F., AND CLERF, L. H.: Congenital Anomalies of the Alimentary Tract, with Special Reference to Congenitally Short Esophagus. Am. J. Roentgenol. **33:** 657–669, 1935.

8. MATHIEU, A., AND GOLDSMITH, H. E.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula. Am. J. Surg. **22:** 233–238, 1933.

9. PLASS, E. D.: Congenital Atresia of Esophagus with Tracheo-esophageal Fistula; Associated with Fused Kidney. Case Report and Survey of the Literature on Congenital Anomalies of the Esophagus. Johns Hopkins Hosp. Rept. **18:** 259–286, 1919.

10. ROSENTHAL, A. H.: Congenital Atresia of Esophagus with Tracheo-esophageal Fistula: Report of 8 Cases. Arch. Path. **12:** 756–772, 1931.

11. SOLIS-COHEN, L., AND LEVINE, S.: Congenital Atresia of the Esophagus with Tracheobronchial Fistula. Am. J. Dis. Child. **51:** 1119–1125, 1936.

12. STRONG, R. A., AND CUMMINS, H.: Congenital Atresia of Esophagus with Tracheo-esophageal Fistula. Am. J. Dis. Child. **47:** 1299–1309, 1934.

13. TUCKER, G., AND PENDERGRASS, E. P.: Congenital Atresia of the Esophagus: New Diagnostic Technic. J. A. M. A. **101:** 1726–1727, 1933.

14. VOGT, E. C.: Congenital Esophageal Atresia. Am. J. Roentgenol. **22:** 463–465, 1929.

Naegele Pelvis: Study of a Case in Labor[1]

RICHARD TORPIN, M.D.

Augusta, Georgia

INASMUCH AS THE origin of Naegele's pelvic deformity is still in question, and because of its rarity, presentation of a case seems justified.

In 1929, Williams (1) described a patient who died from rupture of the uterus following version and extraction delivery of her seventh child, which was probably 500 grams larger than any of the others, all of which, being small, had been born spontaneously. In regard to the etiology he wrote:

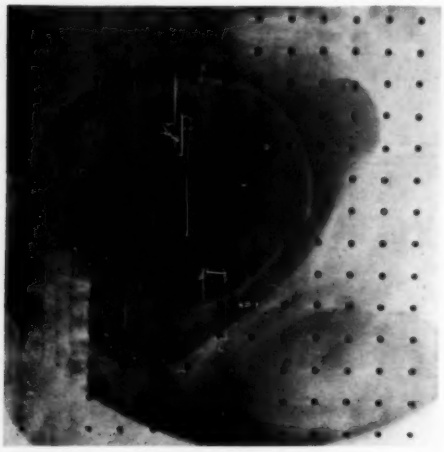


Fig. 1. Centimeter grid roentgenogram of the pelvic inlet, showing the molded compressed fetal head somewhat below engagement.

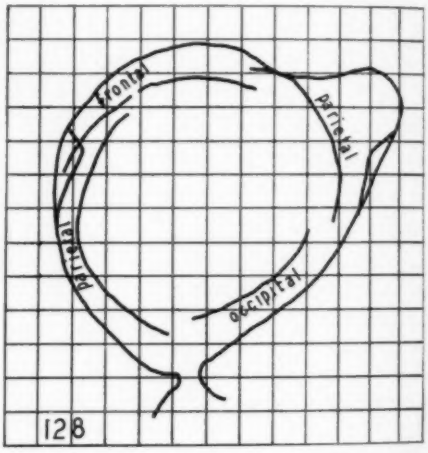


Fig. 2. Pelvigram (on centimeter scale) of pelvic inlet and overlapping bones of fetal head, as labeled. All measurements exact.

"The description of the morphology, in Naegele's original manner, of the obliquely ovate pelvis was so masterly that nothing has since added to it. As is well known, Naegele attributed the deformity to a congenital defect involving one ala of the sacrum with resulting imperfect development of the sacral portion of the sacroiliac joint. Furthermore, when the individual began to move about, the body weight would in great part be transferred to the femur on the affected side, with the result that unusual pressure would be exerted upon the abnormal joint, and that the irritation induced thereby would eventually lead to ankylosis. His original publication was followed by a considerable literature, and all of the earlier contributions were confirmatory of his point of view. Indeed, it was not until 1861 that any scepticism developed, when Thomas of Leyden pointed out that in at least a certain proportion of obliquely ovate pelves the essential feature lay in the destruction and subsequent fusion of an originally normal sacrum as the result of inflammatory disease, rather than in a primary defect in development.

"Since then the discussion has continued, and reached its culmination in 1900, when Breus and Kolisko in their monumental work on deformed pelves stated that the condition is always the result of inflammatory disease, whose existence can generally be elicited from the history of the patient, and particularly from the presence upon the external sur-

[1] From the Department of Obstetrics and Gynecology, University of Georgia School of Medicine.

Fig. 3. Photograph of lumbar region showing slight deformity. Note that the malformed half of the sacrum is on the reader's left in this illustration but on the right in all of the others.

face of her body of cicatrices which indubitably indicate that such disease had existed."

CASE REPORT

M. G., a Negro primipara, aged 18, of slender constitutional type, was sent into the hospital from the outpatient delivery service, deep in the second stage of labor. As is routine in such cases, a roentgenogram was made of the pelvic inlet by a modified Thoms grid method (2), with the result appearing in Figure 1 and in the pelvigram shown in Figure 2. A diagnosis of Naegele pelvis was made as the cause of dystocia, and the position of the fetal head was believed to be left occipitoposterior, one of the markedly distorted parietal bones being mistaken for the occipital bone. The head was in mid pelvis; the first stage of labor had been thirty-two hours and the second stage five hours and fifty-five minutes.

When examination preparatory to application of forceps was made, it was seen that the presentation was left occipito-anterior, with flattening of the skull in the anteroposterior direction. Kjelland forceps were applied to the parietal bones, and the fetal head was delivered on the third 40-second pull. The infant weighed 5 pounds and 8 ounces and cried spontaneously. The measurements of the head in the distorted molded condition were: biparietal 9.25 cm.; suboccipital bregmatic 8 cm.; frontooccipital 12.5 cm. The biparietal diameter in the roentgenogram was 8.5 cm. and the suboccipital bregmatic, 7 cm., showing evident compression of the head, inasmuch as it was fully engaged in the pelvis. The maternal pelvic measurements were: superior spines 20.5 cm.; iliac crests 25 cm.; intertrochanteric 27 cm.; external conjugate (Baudlocque) 21 cm.; outlet 9 cm. The left iliac crest was 1.5 cm. higher than the right.

Inspection of Michaelis' rhomboid (Fig. 3) showed the dimple on the left side to be deeper and nearer the mid-line than on the right side. The patient gave no history of injury or disease of the pelvis and had no recollection of limping or of any difficulty in walking. She started to walk at nine months of age and stated that she never became tired of walking or dancing, and that no one had ever suspected any pelvic trouble. No scars and no other deformities were noted.

Fig. 4. One of several postpartum roentgenograms of the defective sacrum, none of which shows evidence of old infection.

COMMENT

A careful study of each of several roentgenograms (Fig. 4)[2] revealed no evidence of any injury or inflammatory reaction in the clear-cut views of the sacral bone, of which the left ala apparently was entirely absent, with resulting inward displacement and deformity of the left innominate bone. The perfect regularity of the bony fibers implies that the defect was congenital rather than secondary to an inflammatory process.

This case, then, not only in a study of the defect itself but also in the history, strongly supports Naegele's contention that the deformity is a congenital embryonic defect rather than secondary to

[2] The original film is much clearer than the reproduction.

osteitis, as Thomas, Breus, and Koliska and others contend. A congenital etiology is likewise supported by Williams' case, not only from a study of x-rays in life, but from a study of the pelvis after the death of the patient. The same etiology is suggested by Thoms' (3) patient who had, on the same side of the body, a defect of the kidney with double ureter and double kidney pelvis. She presented no evidence of early disease to account for the deformity of the pelvic bones. Reinberger's (4) case also supports the congenital defect theory in that there was no history of any early illness, and other congenital defects were present—bilateral club feet, double vagina, double cervix, and septate uterus. Taddei's (5) case presented no evidence of early childhood infection and apparently none of other external or internal malformations of the genital tract.

School of Medicine,
University of Georgia,
Augusta, Ga.

REFERENCES

1. WILLIAMS, J. W.: Clinical and Anatomic Description of Naegele Pelvis. Am. J. Obst. & Gynec. **18:** 504–514, October 1929.

2. TORPIN, R., HOLMES, L. P., AND HAMILTON, W. F.: Roentgen Pelvimeter Simplifying Thoms' Method. Radiology **31:** 584–586, November 1938.

3. THOMS, HERBERT: Naegele Pelvis: Summary and Description of Case. Yale J. Biol. & Med. **10:** 513–517, May 1938.

4. REINBERGER, J. R.: Naegele Pelvis with Coincidental Deformities of Genital Tract and Extremities. Am. J. Obst. & Gynec. **25:** 834–839, June 1933.

5. TADDEI, A.: Contributo allo studio del bacino ovalare obliquo de Naegele. Rassegna d'ostet. e ginec. **45:** 567–578, November 1936.

Effect of Roentgen Rays on the Peripheral Nerve of the Rat[1]

ARNOLD H. JANZEN, M.D., and SHIELDS WARREN, M.D.

Boston, Mass.

THE EFFECT OF roentgen rays on the peripheral nerves has practical implications because of the increasing use of large therapeutic doses to which they are incidentally exposed. This study aims at determining what, if any, histologic and physiological alterations result from exposure of nerves to x-ray and radium.

Observations on human nerves have been contradictory and incomplete and therefore cannot easily be evaluated. Some workers have been unable to demonstrate any changes (1, 2), and the degeneration described by others (3) is of uncertain significance, since the conditions were not specified.

The experimental data are somewhat more detailed. A mild degeneration of the neurones of the sympathetic ganglia of the rabbit has been produced by direct exposure to roentgen rays (4). Doses of 1,000 to 4,000 r hindered myelination and finally completely destroyed axones and neurones of the newborn rat (5). Rascanu (6), using the ultramicroscope, described a reversible change in the myelin occurring at the Lantermann incisures of the frog nerve irradiated with roentgen rays.

Several observations have been made on the effect of radiant energy on the excitability of the nerve of the frog. Lazarus-Barlow (7) described an increased excitability of the irradiated frog nerve during the thirty- to fifty-hour life of the preparation. Redfield (8) found with small doses of beta and gamma rays an initial increased excitability of the frog nerve, which was followed by a rapid rise in threshold. The initial decrement of threshold was absent when larger doses were used. Similar findings were described by Rascanu (6). Audiat (9) irradiated the isolated frog nerve in a moist chamber with 300,000 r of 1 Å. wavelength, and described loss of excitability developing in a few minutes to half an hour. This change spread 1 cm. above and below the irradiated segment and was reversible if the nerve was placed in physiological saline solution.

Since we did not find any reports of similar work on the nerves of the mammal, it seemed that such experiments followed by histologic observations might add important information provided the time limitations which obtain with the use of an isolated nerve muscle preparation could be avoided. For this purpose we used the intact right sciatic nerve of the rat. The procedure was kept as constant as possible.

METHOD

Stock white rats weighing 170 to 210 grams each and maintained on a vitamin-rich diet were used. Under nembutal anesthesia both sciatic nerves were isolated *in situ* and kept moist with Tyrode's solution, care being taken to prevent the least trauma. A lead shield 10 × 10 × 0.2 cm. with an opening just large enough for exposure of the right nerve was placed over the animal, and the adjacent tissues were protected by a lead shield (0.1 cm. thick for 4,000 r and 0.2 cm. thick for 6,000–10,000 r) placed under the nerve. The factors of radiation were as follows: 200 kv., 8 ma., no extrinsic filtration, cone 5 cm. diameter, target-nerve distance 15.5 cm. The skin was closed and the animals were killed at intervals: 24, 48, 96, 144, 168 hours; 2, 3, 4, 5, 6, 7, and 8 weeks.

An hour before killing the rat, the nerves were tested for excitability, using break shocks from the secondary coil of an inductorium. The measured current in the primary coil was varied with a resist-

[1] From the Laboratory of Pathology, Collis P. Huntington Memorial Hospital. Accepted for publication in November 1941.

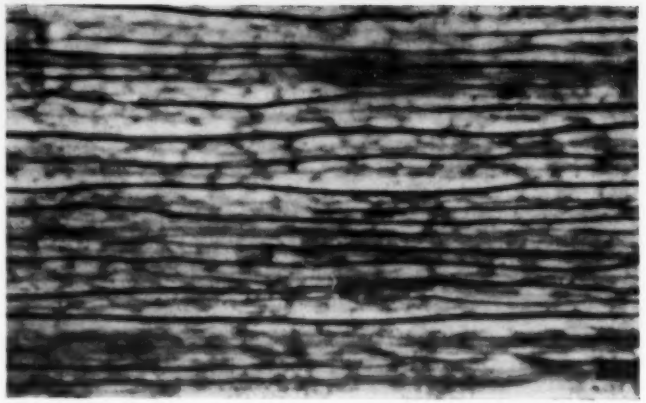


Fig. 1. Sciatic nerve of rat given roentgen ray, 10,000 r, three weeks previous. Silver stain. ×450.

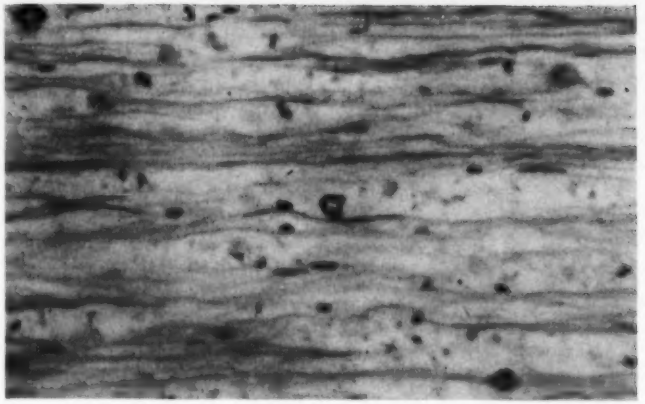


Fig. 2. Sciatic nerve of rat given 1,133 millicurie-hours radon three weeks previous. Silver stain. ×450.

Fig. 3. Same as Fig. 1. Stain for degenerating myelin. ×450.

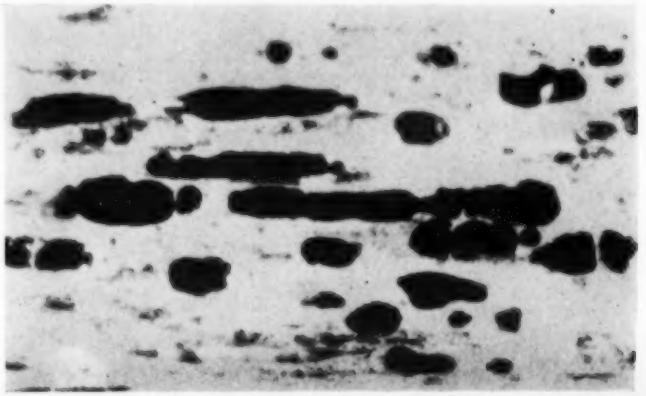


Fig. 4. Same as Fig. 2. Stain for degenerating myelin. ×450.

ance. The secondary coil was placed at 10 cm. and tilted away from the perpendicular, the tilt being measured on a scale. The electrode used had two poles, 1 cm. apart, protected by rubber so that the whole could be incorporated under the skin of the thigh. The Achilles tendon and lower portion of the gastrocnemius were freed, and the leg immobilized at knee and ankle. The free end of the tendon was attached to a myograph and a record of the contractions was made on a revolving drum. Thus each nerve was studied, and since it was found that in any one animal the nerve tested second usually was the more excitable, the right or left nerves of alternate animals were stimulated first. In the early experiments the cathode was placed descending, but in the later groups of animals both cathode descending and cathode ascending stimuli were used; the former presumably measuring excitability of the irradiated segment and the latter measuring conductivity through it.

The animal was then killed and the sciatic and tibial nerve on each side was tied *in situ*, without stretching, onto a cardboard support where it remained until sectioned. The portion of vertebral column and cord supplying the nerve was removed *in toto*. The irradiated portion of nerve was fixed by the method of Davenport (10 *b*) and sections were stained by silver impregnation (10 *a*), hematoxylin and eosin, and Masson's phosphotungstic acid-hematoxylin. The tibial nerve was fixed in 10 per cent formalin and stained by the chlorate-osmic acid (11) method. Two segments of cord were taken: the caudal portion (L-1 and 2) was fixed and stained by Davenport's (10 *a*) method, sections being taken through the spinal ganglia; the cephalad portion (D-11 and 12) was fixed in 10 per cent formalin and sections taken through ganglia and cord were stained with cresyl violet.

RESULTS

None of the rats limped or showed any inability to use the hind legs except for slight reduction in activity for a few days following the operative procedure.

There was little or no difference between the irradiated and the control nerve in excitability to cathode descending break shocks. In some instances the irradiated nerve was five to ten times more sensitive than the control, but in almost as many other rats the control was more excitable than the treated nerve. The variations seemed to depend on which side was stimulated first, and on the cleanness of the nerve sheath dissection.

When a break shock cathode ascending was used, there was still no significant difference noted between irradiated and control nerves. In all but three instances the stimulus needed with cathode ascending was less than that needed with cathode descending to produce both a minimal and a maximal muscle twitch. This was true of the irradiated as well as the control nerves.

Histologic study likewise showed no differences between the control and the irradiated nerves, their ganglia, or anterior horn spinal cord neurones. The irradiated portion of the nerve showed intact axones and there was no alteration in the number of Schwann cells. Myelin stain of the distal nerve segment showed normal lipoid droplets. Occasional sheaths of both control and irradiated nerves stained black, but since these sheaths remained homogeneous, this change was interpreted as artefact.

The sensory ganglia had for the most part intact cell outline, Nissl's granules, and nucleus. In rare cells the nuclei were eccentric with scanty Nissl's substance, but this was found irregularly and on both normal and control sides so that it is impossible to say that the changes were due to the effect of roentgen rays. Motor neurones in the cord were unchanged.

Four additional groups of animals were irradiated as controls for different factors which it was felt might alter the reaction. In one group of 3 animals the conditions of the experiment were the same except that the lead shield beneath the nerve was

Fig. 3. Same as Fig. 1. Stain for degenerating myelin. ×450.

Fig. 4. Same as Fig. 2. Stain for degenerating myelin. ×450.

eliminated and the nerve was exposed without being isolated. This allowed any possible influences from the irradiated adjacent tissue to take effect and at the same time was a control for the scatter from the surface of the shield. The nerves of these rats showed no changes of any sort.

A second group of 3 rats was given 10,000 r to a 1 cm. area of the right leg along the course of the sciatic nerve. The animals were killed at 1, 7, 18 days. The last one showed ulceration of the skin. None of them limped and none showed physiological or histological nerve changes, nor were the neurones abnormal.

TABLE I: BREAK SHOCK CATHODE DESCENDING STIMULI NEEDED TO PRODUCE MAXIMAL AND MINIMAL MUSCLE CONTRACTIONS IN RATS TREATED WITH 10,000 R X-RAY

Time after	Maximum		Minimum	
Irradiation	Irradiated	Control	Irradiated	Control
24 hours	1,032 ma.	1,060 ma.	696 ma.	798 ma.
48 hours	850	1,056	660	660
96 hours	1,000	584	695	158
144 hours	1,245	1,360	1,030	1,054
168 hours	990	990	614	745
2 weeks	850	1,090	290	850
3 weeks	460	880	154	360
4 weeks	800	1,650	350	1,096
5 weeks	850	850	380	614
6 weeks	920	900	614	500
7 weeks	298	1,140	100	900
8 weeks	620	1,250	196	1,000

In another group of normal animals the previously untouched sciatic nerve was tested for excitability and sections were taken of the nerves and cords. These served as controls for the normal left sciatic nerves of the irradiated animals, and they all proved to be similar.

A last group of three rats[2] had 8.5 to 13 millicuries of radon gold seeds planted in the left psoas muscle approximately 1 cm. lateral to the fourth lumbar vertebra and about 0.4 to 0.5 cm. from the sciatic nerve roots and trunk. They received from 1,200 to 1,600 millicurie hours of gamma radiation. For purposes of rough comparison, by the method of White and Failla (12) we calculated this to represent approximately 75,000 to 100,000 r as measured by ionization in air. On the irradiated side the nerve trunks and distal nerve segments were pale and flabby and the thigh muscles wasted. Stimulation of the left sciatic with the secondary coil of the inductorium completely over the primary coil failed to elicit a response, though the right leg responded normally. Osmic acid stain showed fragmentation of the myelin and large fat droplets. In a silver impregnation, only a few axis cylinders were seen to be intact, and the connective-tissue stain showed a marked disorientation of the neurokeratin network, increase of Schwann cells, and infiltration of lymphocytes. The neurones of the right dorsal root ganglia appeared fairly normal, but on the left side occasional cells with scanty Nissl's substance, eccentric nucleus, and chromatolysis were seen. The anterior horn motor neurones did not appear normal in all instances but the variations on both right and left sides were so inconstant as to preclude definite conclusions. This might possibly be accounted for by the supposition that the motor neurones had sufficient time to recover from the changes produced by distal nerve injury.

The size of field irradiated is known to be important in determining the degree of change produced by a measured amount of roentgen radiation on any square centimeter of tissue exposed. The criticism may rightly be made that a piece of tissue $1 \times 0.2 \times 0.2$ cm., such as the sciatic nerve, will not absorb enough radiant energy to be damaged. To meet this criticism, we irradiated the 3 rats with the nerve unexposed and the adjacent tissue not shielded, to see if the presence of surrounding irradiated tissues would produce greater changes in the nerve. Even after eighteen days, although the skin had been ulcerated for a week, no changes were noted in the nerve. This is probably due to two factors: the resistance of the nerve tissue and its independent blood supply.

[2] Available for study through the kindness of Dr. Nathan Friedman, Littauer Fellow in Pathology.

CONCLUSION

Exposure of the peripheral nerves of the rat to roentgen rays in doses of 4,000, 6,000 and 10,000 r generated at 200 kv. produced no demonstrable histological or physiological changes.

Complete degeneration of nerve and some evidence of degeneration of the sensory neurones resulted from exposure to 1,200–1,600 millicurie hours of gamma radiation.

Nerve tissue is extremely resistant to radiation.

Huntington Memorial Hospital
Boston, Mass.

REFERENCES

1. KANOKY, J. P.: Action of the Roentgen Ray on Nerve Tissue. Am. J. Dermat. & Gen.-Urin. Dis. 11: 327, 1907.

2. GRIFFITH, J. Q., JR., AND PENDERGRASS, E. P.: Study of the Effect of Irradiation upon the Lumbar Sympathetic Ganglia in Rats. Radiology **23:** 463–465, 1934.

3. MASUMOTO, K.: Über den Einfluss der Radiumstrahlen auf die peripheren Nerven der Haut. Mitt. a. d. med. Akad. zu Kioto **12:** 305–307, 1934.

4. SUZUKI, S. Experimental Investigation of the Effect of Roentgen Rays upon the Vegetative Nerve: Histological Investigation of Effect of Irradiation of Inferior Mesenteric Ganglion on Female Genitals. Jap. J. Obst. & Gynec. **14:** 410–415, 1931.

5. LEBOUCQ, G.: Action des rayons X sur la formation de la myéline chez le rat blanc. Rev. belge sc. méd. **6:** 383–387, 1934.

6. RASCANU, V., DOROGAN, D., AND KAPRI, M.: L'action des rayons ultraviolets et des rayons X sur la structures des fibres nerveuses myéliniques; la signification fonctionnelle de la myéline. Compt. rend. Soc. de biol. **129:** 83–88, 1938.

7. LAZARUS-BARLOW, W. S., AND DUNBAR, T. J. D.: Experiments on the Action of Alpha, Beta, and Gamma Radiations of Radium on the Muscle-Nerve Preparations of the Frog. Arch. Middlesex Hosp., London **30:** 17–46, 1913.

8. REDFIELD, E. S., REDFIELD, A. C., AND FORBES, A.: Action of Beta Rays of Radium on Excitability and Conduction in the Nerve Trunk. Am. J. Physiol. **59:** 203–221, 1922.

9. AUDIAT, J.: Action du rayonnement X sur les paramètres d'excitabilité du nerf. Compt. rend. Soc. de biol. **110:** 365–367, 1932.

AUDIAT, J., AUGER, D., AND FESSARD, A.: Etude des potentiels d'action des nerfs soumis au rayonnement X. Comparaison avec l'action des rayons ultraviolets. Compt. rend. Soc. de biol. **116:** 880–883, 1934.

(10) (*a*) DAVENPORT H., A., MCARTHUR, JANET, AND BRUESCH, S. R.: Staining Paraffin Sections with Protargol: 3. Optimum *p*H for Reduction. 4. A Two-Hour Staining Method. Stain Technol. **14:** 21–26, 1939.

(*b*) BANK, E. W., AND DAVENPORT, H. A.: Staining Paraffin Sections with Protargol: 5. Chloral Hydrate Mixtures with and without Formamide for Fixing Peripheral Nerves. Stain Technol. **15:** 9–14, 1940.

11. SWANK, R. L., AND DAVENPORT, H. A.: Chlorate-Osmic-Formalin Method for Staining Degenerating Myelin. Stain Technol. **10:** 87–90, 1935.

12. WHITE, T. N., MARINELLI, L. D., AND FAILLA, G.: Measurement of Gamma Radiations in Roentgens. Am. J. Roentgenol. **44:** 889–903, 1940.

An Electric Model of an X-Ray Machine[1]

MARVIN M. D. WILLIAMS, PH.D.

Division of Physics and Biophysical Research
Mayo Foundation, Rochester, Minnesota

IT IS WELL known that the quantity and quality of radiation emitted by an x-ray tube vary with the wave forms of both the voltage applied to the tube and the current flowing through the tube. The voltage wave form depends primarily on the type of rectifying circuit used and to a lesser degree on the characteristics of other parts of the x-ray machine, such as the transformer, the primary circuit, and the x-ray tube. The current wave form depends, of course, on the voltage wave form, but is equally dependent on the characteristics of the x-ray tube unless the rectifying circuit supplies constant potential. Hence measurements of peak volts applied to the x-ray tube and of the average current flowing through the tube, which are the common factors measured, give no idea of the quantity and quality of the radiation which will be emitted from the tube.

It would seem, therefore, to be of some value to radiologists to know the general type of wave forms produced by the more commonly used rectifying circuits and the changes in the wave forms produced by alterations which may be made on x-ray machines, as well as the effect of various wave forms on the quantity and quality of the emitted radiation. This, of course, requires some knowledge of the action and function of the various parts of a machine and how they are connected together. This knowledge can be obtained from books and lectures, but many people learn and remember better from doing than by reading or hearing alone, as is evidenced by our supplementing lecture courses with laboratory work.

The training of radiologists in the physical principles involved in their specialty should include some laboratory work as well as lectures and reading. As part of the laboratory work it would be desirable to have them study the construction of different kinds of x-ray equipment and determine the effect of various types of rectification on the quantity and quality of x-rays produced. In most institutions this is not a practical procedure, because different types of equipment are not available and the compactness of most equipment makes it difficult, if not impossible, for one unfamiliar with such circuits to learn much about the construction of an x-ray machine and the relationships and functions of the various parts. The effect of various rectifying circuits on the radiation produced by an x-ray tube can be studied only by using x-ray equipment having the different circuits, but a knowledge of the voltage and current wave forms supplied by various types of circuits makes it easier to understand why the radiation output varies.

In order to aid students in understanding the construction and operation of x-ray machines, several methods have been used. Wiring diagrams have been drawn in varying degrees of simplicity. Parts of machines have been obtained, shown, and occasionally operated in circuits of varying degrees of completeness. Models have been built with colored strings to indicate the various circuits. It was from seeing some of these string models that I obtained the idea of building a model from radio parts, which would function electrically much like an x-ray machine except that the voltages generated would be low, and which could be altered easily to demonstrate many of the commonly used rectifying circuits. The use of an oscillograph with the model would show the wave forms of the voltage and current in any part of the secondary circuit.

[1] Shown in the Scientific Exhibits at the Twenty-sixth Annual Meeting of the Radiological Society of North America, Cleveland, Ohio, Dec. 2–6, 1940.

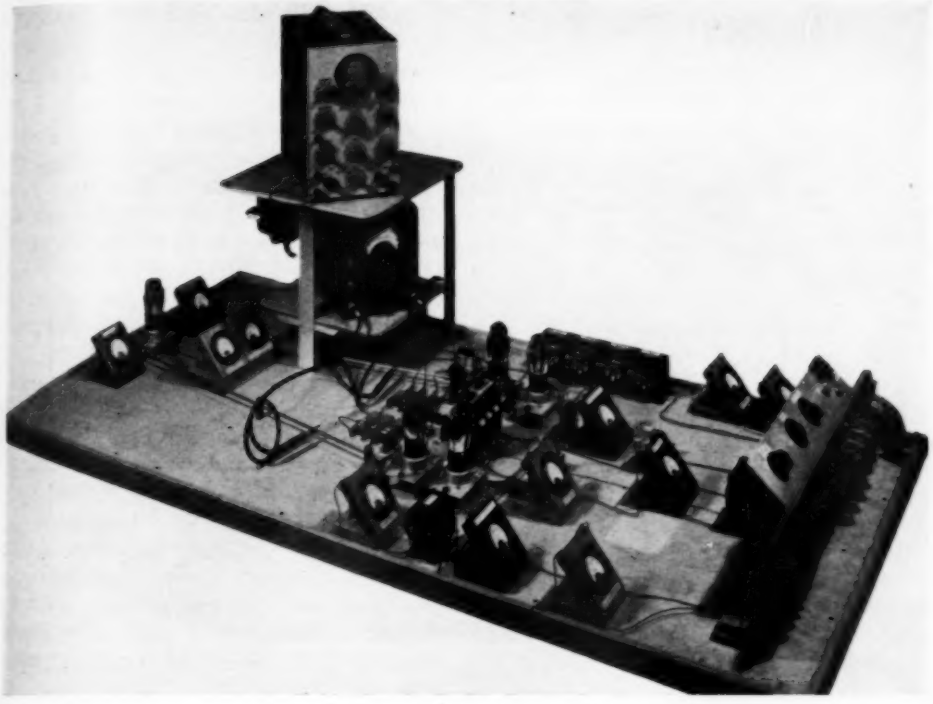


Fig. 1. Electric model of an x-ray machine.

At the time that this model was built, I was unaware that anything similar had ever been constructed. Later it was learned that a few other people had built sets with more or less the same ideas in mind. Hence, although this set was built without knowledge of the others, no priority is claimed. In view of the many requests, however, which have been received for diagrams, pictures, and information concerning the set, there would appear to be sufficient interest to warrant a description of the model, and it is to be hoped that others will improve on it. The set is not meant to represent any particular x-ray machine or to include all the controls and accessories that may be found on x-ray equipment. The purpose is to show the essential and most commonly used parts of a machine, to keep the entire set relatively uncomplicated, to allow study of the function and action of as many parts as possible, and to have the secondary or high-tension circuit as flexible as possible.

Figure 1 is a picture of the set and Figure 2 shows the basic wiring diagram. The board on which the set is mounted is 5 feet (152 cm.) long by 2 1/2 feet (76 cm.) wide. The relative positions of most of the equipment are the same in the two figures, except that the positions of meters A_5 and V_5 are interchanged, and meters A_1 and V_1 and switch S_1 are to one side of the board. The fuses (F's), connections to the autotransformer, and all the resistances (r's and R's) except the rheostat r_5 are mounted on the front of the vertical panel. The autotransformer is mounted just behind this panel. The rheostat and voltage control, switches S_2 and S_3, the pilot light (PL), and filament control (r_5) are mounted on the sloping panel above the vertical panel. The oscillograph and the control box for it are mounted on shelves, and a switch below the upper shelf changes

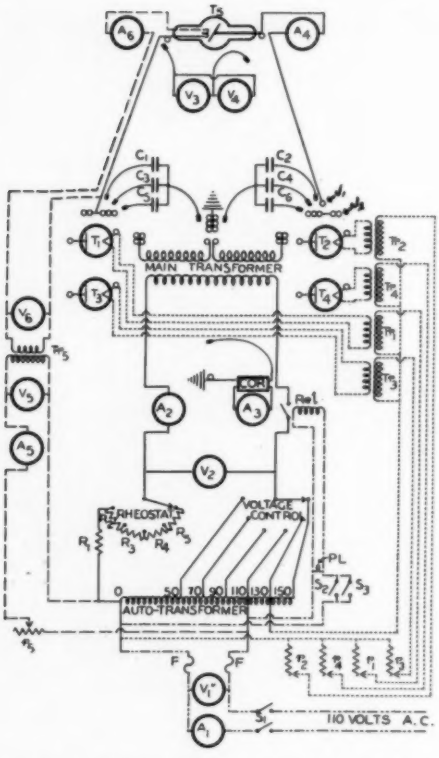


Fig. 2. Diagram of permanent wiring showing the complete wiring diagram of the primary, control, and x-ray tube and valve tube filament circuits. Connections between the secondary of the transformer and the x-ray tube for the various rectifying circuits are shown in Figures 3 to 6.

the connections so that either voltage or current wave forms can be shown on the oscillograph. The use of wires of different colors enables each circuit to be traced easily. The wiring shown in Figure 2 is all permanent and includes the primary circuit for the main transformer, the control circuit, the x-ray tube filament circuit, and the circuits for the filaments of the four valve tubes.

In order to show current and potential relations, many more meters were used than are commonly found on x-ray machines; those which are commonly found were mounted on dark-colored supports. The alternating and direct current voltmeters, V_3 and V_4, can be connected across the x-ray tube when a rectified potential is applied to the tube, and with the oscillograph can show the difference between effective, average, and peak volts. These two meters are omitted in the drawings of the rectifying circuits of Figures 3 to 6. Milliammeter A_3 is connected to a grounded point in the secondary circuit. In some circuits the current through this meter is unidirectional and in other circuits it is alternating. In order to use only one meter, instead of an A.C. and a D.C. meter, a full-wave copper oxide rectifier (*COR*) is used for this meter.

Probably many types of radio tubes could be used for the rectifiers and x-ray tube. Type 80 tubes have proved satisfactory both for rectifiers and for the x-ray tube. The two plates of these tubes are connected together.

Many of the voltages generated are too high to be applied directly to the deflection plates of the oscillograph; hence a voltage divider is used. This divider consists of two 200,000-ohm resistances in series with a 100,000-ohm resistance, the mid-point of which is grounded. The plates of the oscillograph are connected across the 100,000-ohm resistance; hence the deflection on the oscillograph is due to one-fifth of the total voltage. The 100,000-ohm resistance is contained inside the control box (1), and the two 200,000-ohm resistances are connected to the ends of two cables with clips on the other ends of the resistances for connecting to the desired points in the circuit. These clips can be attached to any two points of the circuit between which it is desired to observe the potential wave form on the oscillograph.

To observe the wave form of the current in any part of the circuit, it is necessary to connect the oscillograph across a non-inductive resistance, and thus one really observes the wave form of the voltage drop across this resistance. In order to have the voltage drop across this resistance large enough to produce observable deflections when applied directly to the plates of the oscillograph, it would be necessary to use a resistance of the order of 50,000 to 100,000 ohms, since the currents

are generally only a few milliamperes. Such a large resistance would alter the characteristics of the circuit seriously. Hence a small resistance, approximately 25 ohms, is used, and the potential drop across this resistance is applied to the amplifying system of the oscillograph. The interpretation of what is observed may be rather difficult at times, since the amplifier responds only to variations in potential (constant currents do not produce any deflection). This small resistance generally is connected between the points J_1 and J_2 (Fig. 2) for showing the x-ray tube current, although it may be connected into any part of the high-tension circuit.

The primary circuit for the main transformer includes one voltage control for varying the autotransformer connections, a relay switch, the primary of the main transformer, a rheostat control, a voltmeter (V_2), and an ammeter (A_2). Most machines have a major and a minor voltage, or autotransformer, control, but one such control shows the principle and is simpler than two. The relay switch is generally a two-pole instead of a one-pole switch. The rheostat seldom is included in diagnostic equipment, and in modern therapeutic equipment it generally activates a switch in such a manner that the relay will not function until all the resistance is included in the circuit. The ammeter seldom is found on an x-ray machine. An overload circuit breaker, which is nearly always found, was omitted chiefly because of the great variation in current that occurs owing to the different rectifying circuits employed; it easily could and probably should be included.

The control circuit includes two switches (S_2 and S_3) in parallel, a pilot light (PL), and the coil of the relay. The two switches illustrate the connections for a hand switch and a timer, or a hand switch and a foot switch; a logical change in this circuit would be to include a second switch in series with one of these to illustrate, for example, the necessity of closing the "foot switch" on the control panel before the real foot switch can function. Another switch in series with both of these switches could be added to illustrate the use of switches such as those on doors to treatment rooms, which prevent the energizing of the main transformer when the doors are open. Additional switches on the relay often are used to activate such devices as exposure counters and clocks for timing the life of tubes.

The x-ray tube filament circuit includes a rheostat (r_5), two voltmeters (V_5 and V_6), two ammeters (A_5 and A_6), the filament transformer (Tr_5), and the filament of the x-ray tube (T_5). A variable choke coil nearly always is used instead of a rheostat, and if a small one could be purchased or made it would be preferable, although either one is suitable for controlling the filament temperature. Some machines, particularly therapeutic equipment, do not have any meters in this circuit, although nearly all diagnostic equipment includes one or both of the ammeters. Since this is a simple transformer circuit, the four meters were included, in order that relations between current and voltage in the primary and secondary of a transformer circuit could be observed.

The valve tube filament circuits are essentially similar to the x-ray tube filament circuit. Each circuit contains a rheostat (r_1, r_2, r_3, and r_4), a transformer (Tr_1, Tr_2, Tr_3, and Tr_4), and the filament of a valve tube (T_1, T_2, T_3, and T_4). Occasionally a machine has voltmeters or ammeters in these circuits, but they were omitted because of the similarity to the x-ray tube filament circuit. Often a machine using four valve tubes uses only three filament transformers, operating two of the valve filaments from one transformer; an attempt to do this would have limited to some extent the flexibility of this set, and therefore a separate transformer was provided for each tube. The range of x-ray tube currents available here does not warrant changing valve filament temperatures; hence the rheostats are set so that there are approximately 110 volts across the primaries of the transformers. This

voltage could be obtained by connecting to the 110-volt tap of the autotransformer and omitting the rheostats; however, all machines using valve rectification have rheostats for adjusting their filament currents.

Not shown in the diagram is a ground wire attached to the indicated grounding connections, to the cases of all transformers, and to all other metal parts which are not part of the electric circuits. This ground wire is attached to a water pipe or other good ground connection.

Connections between the secondary of the main transformer, the x-ray tube, the valve tubes, the condensers, and the "grounder milliammeter" (A_3, through the copper oxide rectifier *COR*), are made by plugging into phone tip jacks (for example, circles at ends of transformer secondary in Figure 2) short pieces of wire provided with phone tips on their ends (for example, ends of wires connected to condensers in Figure 2). This arrangement allows connections to be made or changed quickly in order to show many different types of rectification. Practically all the more commonly used types of rectifying circuits can be set up. Although it has not been done on this set, it should be easy to add a small synchronous motor so that mechanical rectification could be shown. Figures 3 to 6 are diagrams of seven circuits which can be shown easily. When any of these circuits is used on x-ray equipment, resistances or choke coils, which are not shown here, may be used for various purposes; they are not, however, fundamental parts of the circuits. Each of the six condensers used has the same capacity; it would be of some advantage to have three different sizes, probably a smaller and a larger size than that used here.

The actual voltages and currents, and their wave forms, obtained from any x-ray equipment, will depend to some extent on the electric characteristics of the transformers and other equipment in the circuits. The various parts of the machine probably will be designed to operate together and their characteristics will be so chosen that they operate efficiently at the current and voltage values for which the machine is designed. Various auxiliary equipment may be added to increase efficiency or to eliminate undesirable effects. Obviously such considerations cannot be included in a model set of this type. These problems are of primary interest and concern to the electrical engineer who designs the equipment and not to the radiologist, who is primarily interested in the general principles involved. The purpose of this set is to illustrate primarily those general principles with which the radiologist should be familiar.

Observations which can be made for any of the rectifying circuits, and which are in general the same for all the circuits, are the effect of varying the voltage control (autotransformer setting) on the primary voltage (V_2), the x-ray tube voltage, and x-ray tube current; the effect of varying the amount of resistance in the primary circuit (controlled by the rheostat) on the primary and x-ray tube voltages; the effect of varying the x-ray tube current on the primary current (A_2) and on the primary and x-ray tube voltages and the manner in which these effects depend on the amount of resistance (setting of rheostat) in the primary circuit; the difference between the prereading and load-reading of the potential (V_2) applied to the primary of the main transformer, and how this difference depends on the magnitude of the x-ray tube current and on the amount of resistance in the primary circuit.

In Figures 3 to 6 the left-hand diagrams are simple sketches of the high voltage circuit. The diagrams on the right illustrate the necessary connections on the model set to duplicate the circuit shown on the left. Since the primary, control, and x-ray and valve filament circuits remain unchanged for all these circuits, they are not shown in these figures.

Figure 3*a* represents what is commonly called a self-rectified circuit. This designation may be misleading, since it does not specify what is rectified. The voltage applied to the x-ray tube is not recti-

fied but is alternating, although the current is rectified since the x-ray tube allows current to flow only in one direction, or during only half of the voltage cycle. The wiring of this circuit can be altered, and often is in practice, by grounding either end of the transformer secondary instead of its mid-point. It can be observed that the useful peak voltage (half cycle when current is flowing through the tube) is less than the inverse or blocked peak voltage, and that this difference increases with an increase in tube current or with an increase in the amount of resistance in the primary circuit. The effect of using shockproof cables, which, from the electric standpoint, is the same as putting a condenser between each end of the tube and ground, can be shown by connecting in one or more condensers from each set (as shown by the dotted lines in Figure 4*a*). This will be observed to have little effect on the x-ray tube voltage and current (A_4) but will increase the primary current (A_2) and the transformer current (A_3) greatly. If, however, the end instead of the center of the transformer is grounded, and meter A_3 is connected be-

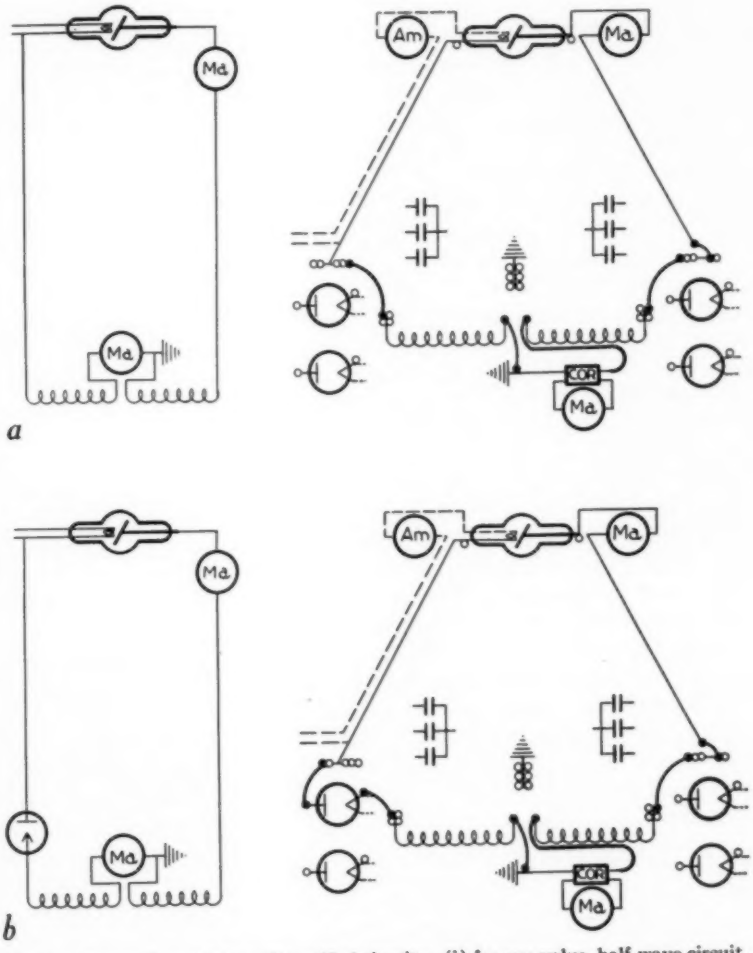


Fig. 3. Connections (*a*) for self-rectified circuits; (*b*) for one-valve, half-wave circuit.

tween the end of the x-ray tube and ground, the addition of the condensers has no more effect on A_2 than it does on A_4, but still greatly increases the reading of A_2. This illustrates an error which may be introduced in measuring tube current on shockproof equipment if the milliammeter is connected so as to indicate the total current in the grounded circuit of the transformer. The effect that the condensers have on the current in the transformer can be shown on the oscillograph by using the small resistance for the connection between the transformer and ground.

Figure 3*b* represents a single-valve rectifying circuit, in which the valve tube as well as the x-ray tube prevents a current from flowing in the wrong direction, and each tube blocks half of the inverse voltage. For this circuit the useful peak voltage is always much greater than the inverse peak voltage across the x-ray tube. Except for half of the inverse voltage being blocked by the valve tube, this circuit is essentially the same as that in Figure 3*a*. The effect of shockproof cables, however, (condensers) is quite different. Since one of the cables (condensers) is prevented, then, by the valve from discharging through the transformer, it discharges through the x-ray tube and hence increases the x-ray tube current, reduces its inverse voltage, and distorts the useful voltage wave. The cable on the opposite side can still discharge through the transformer and hence produces the same effects as in the self-rectified circuit. Cables can be prevented from causing an erroneous reading on A_3 if this meter is connected to the half of the transformer which also is connected to the valve. The valve can be placed between the mid-point of the transformer and ground, an arrangement which has the advantage that the filament circuit for the valve tube is at ground potential. If, instead of grounding the center of the transformer, the end opposite the one connected to the valve is grounded, no inverse potential is applied to the x-ray tube and full half-wave rectification is obtained by the one valve; in this circuit shockproof cables do not produce an erroneous reading on meter A_3 but do distort the voltage wave applied to the x-ray tube and increase the current.

Figure 4*a* represents a two-valve rectifying circuit which gives half-wave rectification. The two valves prevent any inverse potential from being applied to the x-ray tube. If rather large condensers are added to this circuit (shown by dotted lines), nearly constant potential will be applied to the x-ray tube; but if the condensers are small, such as shockproof cables, the voltage wave form applied to the x-ray tube will be a much distorted half wave which in general will reduce the output of radiation. Without condensers each valve must block half of the inverse potential; but as condensers are added, the magnitude of the inverse potential across each valve tube increases so that, if constant potential is obtained, the inverse potential across each valve is approximately equal to the total peak voltage developed by the transformer. Even if the x-ray tube current is kept constant, the value of the peak current through the valve tubes increases as the size of the condensers is increased, but as the peak value of the current increases, the length of time during which current flows through the valves decreases; the quantity of current which flows through the valves remains constant and equal to the quantity of current which flows through the x-ray tube. Without condensers the wave form of the current through the valves is the same as that through the x-ray tube. For any one size of condensers used, the x-ray tube voltage wave form depends very much on the x-ray tube current; the smaller the current, the more nearly is constant potential obtained.

Figure 4*b* represents a four-valve rectifying circuit which gives full wave rectification. This is the circuit probably most commonly used on machines for diagnostic work. When this type of rectification is used on a machine for therapy, condensers are sometimes added (as shown by dotted lines in the figure) so that the x-ray tube

will be supplied with nearly constant potential. The use of shockproof cables will produce a distortion in the voltage wave but, since, as compared with the two-valve, half-wave circuit of Figure 4*a*, much smaller condensers are required to obtain the same degree of constancy of potential on the x-ray tube, the use of shockproof cables generally tends to increase the output of radiation. Regardless of whether or not condensers are used, the valve tubes must withstand an inverse potential, during the half cycle during which they do not pass current, equal to the peak potential developed by the transformer, which is the same as the peak potential applied to the x-ray tube. The peak value and duration of flow of current through the valves as compared with those through the x-ray tube vary with the amount of capacity (condensers) in the circuit in the same general way as they do for the two-valve, half-wave circuit shown in Figure 4*a*.

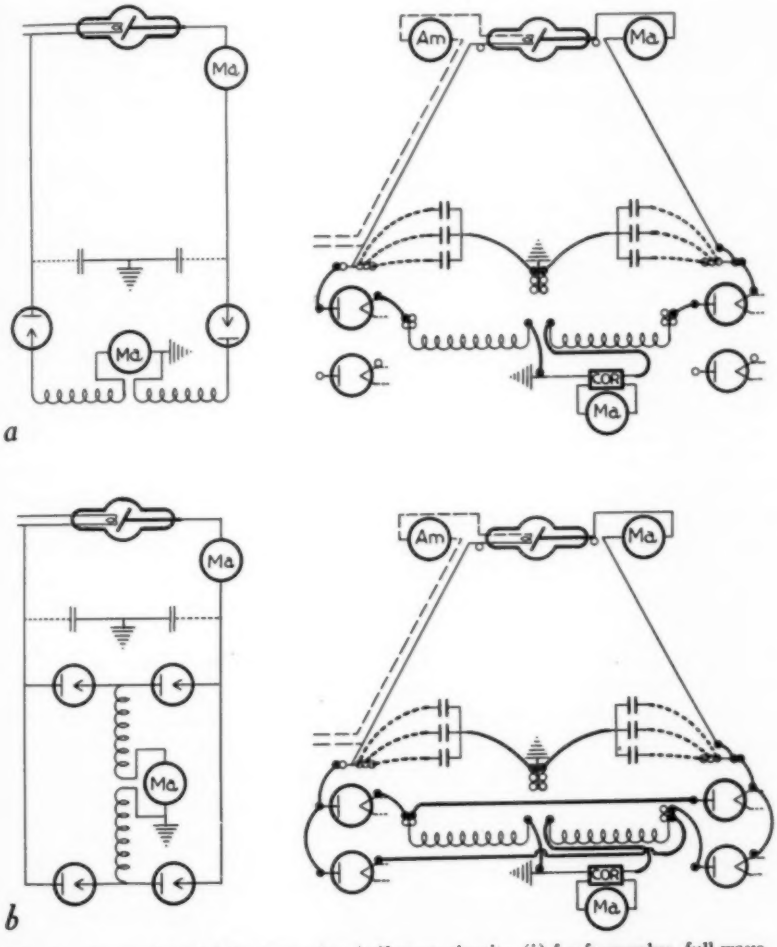


Fig. 4. Connections (*a*) for two-valve, half-wave circuit; (*b*) for four-valve, full-wave circuit.

Figure 5*a* represents a Villard circuit, in which the x-ray tube is supplied with a fluctuating potential. Although generally this circuit is spoken of as a voltage-dou-

bling circuit, the peak potential across the x-ray tube may not be double the peak potential developed by the transformer; how nearly it approaches being doubled depends on the x-ray tube current and the capacity of the condensers. For the same transformer potential the x-ray tube potential increases as the capacity of the condensers is increased or as the current through the x-ray tube is decreased. With the milliammeter A_3 connected as shown in Figure 5*a*, between one of the valve tubes and ground, its reading corresponds with that of the milliammeter (A_4) in the x-ray tube circuit, showing that the average current through the valves is the same as that through the x-ray tube; however, the oscillograph shows that current flows through the valves for a shorter period during each cycle than through the x-ray tube and has a peak value as much as two or three times the peak value of the current through the x-ray tube. If A_2 is connected between either half of the transformer secondary and ground, its reading will be practically double that of A_4; this shows that the power, which is approximately equal to the product of the current and the potential, developed in the transformer is approximately equal to the power used by the x-ray tube; the potential is doubled in one circuit while the current is doubled in the other circuit. It is possible to use a single valve (omitting the milliammeter and ground connection) instead of two valves, but with one valve the inverse potential across the valve is the same as the useful peak potential across the x-ray tube, while with two valves the inverse potential across each valve is only half as great. The maximum potential drop across either condenser is equal to half the peak potential generated by the transformer or approximately a fourth the peak potential across the x-ray tube.

Figure 5*b* represents a La Tour or Greinacher circuit, in which the x-ray tube is supplied with a nearly constant potential equal to approximately twice the peak potential generated by the transformer. As in the Villard circuit (Fig. 5*a*), for a constant transformer potential, the potential across the x-ray tube increases as the size of the condensers is increased or as the current through the x-ray tube is decreased. These same factors, which tend to increase the potential across the x-ray tube, also tend to produce a more constant potential. True constant potential and a potential double that of the transformer peak potential can be obtained only with zero tube current, which of course would not produce any x-rays. As in the Villard circuit, the current through the transformer is twice that through the x-ray tube (as can be shown by connecting meter A_3 between the end of the transformer and ground). Hence, the power developed by the transformer is approximately equal to the power used by the x-ray tube, although it is a voltage-doubling circuit. The valve tubes in this circuit must withstand an inverse potential approximately equal to twice the transformer peak potential or equal to the x-ray tube potential, and the potential across each condenser is approximately equal to the transformer peak potential or half the x-ray tube potential. The current through the valves flows for only a short period during each cycle, the time of current flow depending on the size of the condensers and the x-ray tube current, but the quantity of current flowing through each valve is equal to the quantity flowing through the x-ray tube, the current of which is nearly constant; hence, when current does flow through a valve, its magnitude may be as much as ten or fifteen times the average current through the x-ray tube. If A_3 is connected between one set of condensers and ground, as shown in Figure 5*b*, it reads a little higher than A_4 because it records a part of both the charging and discharging currents of the condensers. In order to obtain the same reading on A_3 and A_4, it is necessary to connect A_3 into the rectifier (*COR*) circuit so that only one half cycle of the rectified current passes through it, the other half cycle being allowed to go to ground without going through the meter.

Figure 6 represents a Witka circuit, in which the potential across the x-ray tube approaches a peak value three times that of the transformer peak potential; often it is referred to as a voltage-tripling circuit. True tripling of the voltage can be obtained only when there is no current through the x-ray tube. For a constant transformer potential the potential on the x-ray tube increases as the size of the condensers increases or as the current through the x-ray tube decreases. The reading of meter A_3 is nearly three times that of A_4, showing that the power developed in the transformer is approximately equal to that used by the x-ray tube. Since the center of the transformer is the only grounded point in the circuit, there does not appear to be any method of connecting a grounded milliammeter into the circuit and having it read the same as meter A_4. The current through the valves, since it flows during only part of each cycle, is greater than that through the x-ray tube, and the valves must withstand an inverse potential equal to twice the transformer peak potential. The potential across the condensers is equal to the trans-

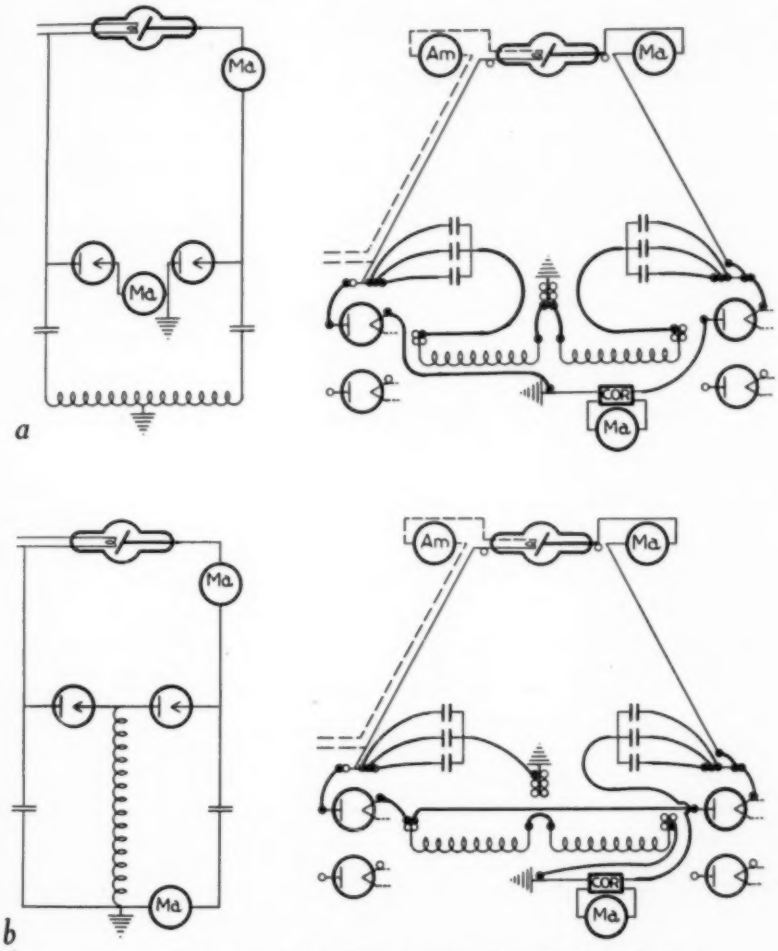


Fig. 5. Connections (*a*) for Villard circuit; (*b*) for La Tour circuit.

former peak potential. This circuit is not often used in America.

The circuits described in the preceding paragraphs represent most of the more commonly used fundamental circuits for x-ray tube potentials up to a few hundred kilovolts. Many variations of these may be set up on this model. By the use of additional condensers, resistances, and inductances, conditions found on almost any circuit can be reproduced fairly well. In attempting to reproduce conditions found on an x-ray machine, it must be remembered that conditions on the model set are not the same as on a real machine.

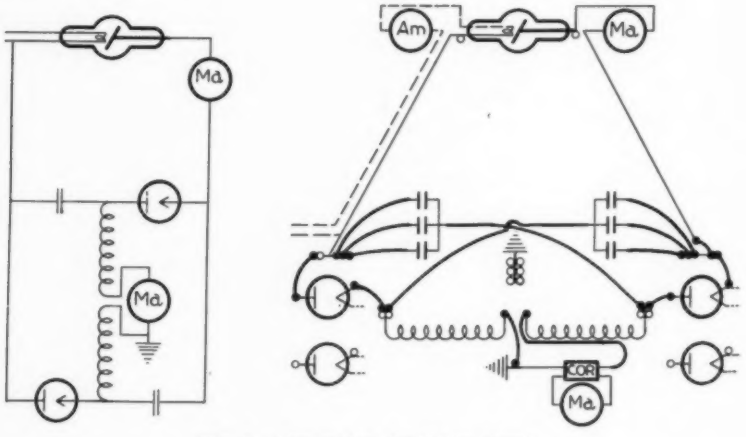


Fig. 6. Connections for Witka circuit.

The electric characteristics of the parts of the model set are different from those on a real machine and the voltages generated on the model set are of the order of a thousandth of the voltages used to generate x-rays. The difference in voltages is particularly important when one is considering the effects of condensers; a very small condenser may produce a large effect on an x-ray machine, while to produce the same effect on the model set, operating with the same x-ray tube current as the x-ray machine but with only one thouandth the voltage, will require a condenser with a capacity one thousand times as great.

EQUIPMENT USED

With the exception of the main transformer, the autotransformer, and the control box for the oscillograph, all the equipment used is standard and can be purchased at any radio store. The two transformers were made to order. The control box for the oscillograph was made first for use in calibrating x-ray equipment with the oscillograph and has been described (1). The total cost of all the equipment, except the oscillograph and control box for the oscillograph, was less than $90. A 3-inch (7.6 cm.) oscillograph has been used because it was available; a 1-inch (2.5 cm.) oscillograph should be satisfactory for small groups. The control box for the oscillograph is not essential, although it offers a convenient means of determining the values of the potentials observed.

The references in the following list of equipment are to Figure 2. The main transformer and autotransformer were specially made without cases. Specifications were as follows:

For main transformer
Primary, 50 to 150 volts, 60 cycle (1 coil)
Secondary, 60 to 180 volts overall at 5 to 30 milliamperes, to be wound in two separate coils

Autotransformer: 60 cycle, 150 volts overall, tapped at 50, 70, 90, 110, and 130 volts. Output sufficient to supply foregoing transformer as well as to provide a load as high as 1 ampere from the 130 volt tap

All the rest of the equipment is standard.

V_1 = V_5 = 0–150 volts A.C.
V_2 = 0–250 volts A.C.
V_3 = 0–500 volts A.C.
V_4 = 0–500 volts D.C.
V_6 = 0–10 volts A.C.
A_1 = A_6 = 0–3 amp. A.C.
A_2 = 0–250 ma. A.C.
A_3 = A_4 = 0–50 ma. D.C.
A_5 = 0–100 ma. A.C.
F = 1.5 amp. 250-volt Littelfuse
S_1 = *DPST* knife switch
S_2 = *SPST* toggle switch
S_3 = Push-button switch (door bell)
Rel = *SPST* normally open relay, 110-volt coil
PL = Neon pilot light
COR = Copper oxide rectifier, full wave, 50-ma. capacity
C_1 = C_2 = C_3 = C_4 = C_5 = C_6 = 400-volt, 1 mfd. condensers (paper)
Tr_1 = Tr_2 = Tr_3 = Tr_4 = Tr_5 = Filament transformers (5 volt, 4 amp. secondaries, 110 volt primary)
T_1 = T_2 = T_3 = T_4 = T_5 = Type 80 tubes (both anodes connected together)
r_1 = r_2 = r_3 = r_4 = 250-ohm 25-watt rheostats (lug adjustment)
r_5 = 2000-ohm 25-watt rheostat (knob adjustment)
R_1 = 25-ohm 10-watt wire wound resistance
R_2 = R_3 = R_4 = R_5 = 150-ohm 10-watt wire wound resistance
Rheostat adjustment = 1 shorting rotary switch (5 circuits)
Voltage control = 1 nonshorting rotary switch (6 circuits)
Small circles (for example, at ends of secondary of main transformer) = Insulated phone tip jacks (38 used)
Plugs on end of wires (for example, leads from condensers) = Phone tips (about 50 used). (An assortment of wires 3 inches to 12 inches (7.6 to 30.5 cm.) long with phone tips on both ends are used for connectors in high-tension system)
3/16-inch (5 mm.) copper tubing used as "high tension" connection from banks of five phone tip jacks (just above T_1 and T_2) to x-ray tube. (Copper tubing on right side ends in separate phone tip jack which usually is connected to the bank of five jacks; this connection is often a 25-ohm non-inductive resistance used to show current wave form on oscillograph)
Solid line = Red wire (main primary and secondary circuits)
Dashed line = Blue wire (x-ray tube filament circuit)
Dash-dot-dash line = Green wire (control circuit)
Dash-2 dots-dash line = Black wire (supply line)
Dotted line = Yellow wire (valve tube filament circuits)

Mayo Foundation, Rochester, Minn.

REFERENCE

1. WILLIAMS, M. M. D.: Two Instruments for Measuring X-ray Tube Voltage. Radiology **38:** 80–83 January 1942.

A Portable Geiger-Müller Counter[1]

ROBERT B. TAFT, M.D.

Charleston, South Carolina

WHILE all of the material presented here is not new, the original circuit was published in a physics journal (1) to which few radiologists have access, and some important changes have since been made.

Fig. 1. Portable Geiger-Müller counter in use; impulses heard in headphones being recorded on a tally counter.

While the principle involved in a counter for field use is the same as that in the more complicated laboratory instrument, the practical design must differ greatly, as simplicity, ruggedness, and freedom from connection to power lines must be maintained without sacrifice of sensitivity. The above requirements appear to be well met in the little instrument described here, as it has been successful in the recovery of much radium, not only by the author but by Dr. George Henny of Philadelphia, Dr. Kenneth Corrigan of Detroit, and others.

As shown in Figure 1, the instrument is completely contained in a steel box measuring approximately 5 1/2 × 6 × 3 inches, which is carried in a canvas case having a pocket for the headphones. Thus the hands of the operator are left free for carrying a stop-watch and tally counter with which to record the number of impulses per minute.

The sensitivity is the same as that of other counters described by the author. "Sensitivity" is arbitrarily defined as the ability to double the normal count when 50 mg. of radium are located at a distance of about 212 feet, this count being directly proportional to the amount of radium and inversely proportional to the square of the distance.

Fig. 2. Instrument in standard steel cabinet. Vibrator adjustment knob on left, switch on right, phone jacks on side.

With a brief description, the diagram (Fig. 3) should be self-explanatory. The old type Ford spark coil normally has one end of the secondary winding grounded to the primary. This end must be re-

[1] Accepted for publication in November 1941.

moved from the primary and brought out by a well insulated lead to the plate and screen of the rectifier tube. This end of the secondary is easily located by fluoroscopy. Proper polarity of the primary to the battery must be determined by trial, since the secondary current is partly unidirectional and, to use the weaker alternation of the cycle, will result in sparking across the tube socket.

The choke coil in series with the filament of the amplifier tube almost entirely prevents the sound of the vibrator in the phones. This choke consists of the core from any miniature transformer, wound with about six ohms (DC resistance) of No. 28 wire.

The vibrator spring should be ground to about half thickness and half width to lower the secondary current. In the first

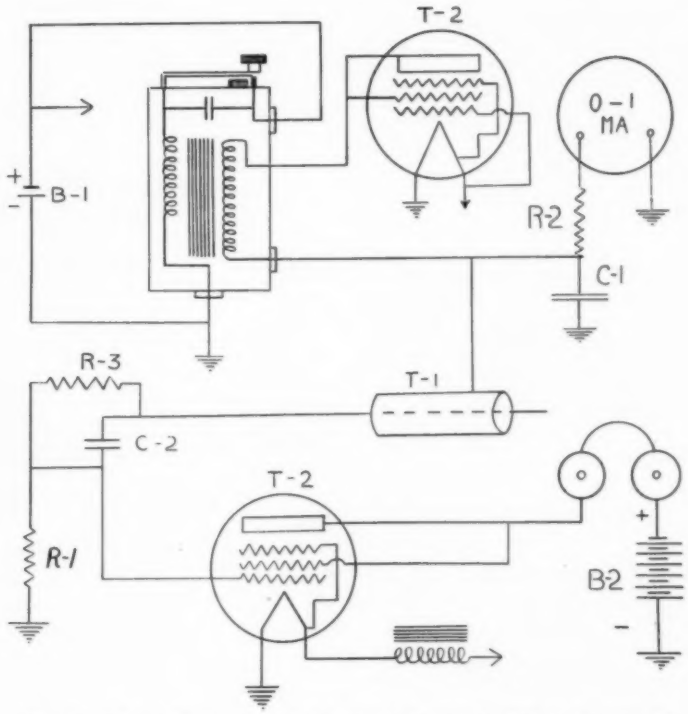


Fig. 3. Wiring diagram. B-1. 1 1/2 volt Everready 742 or Burgess 4F. B-2. 67 1/2 volt Everready 467 or Burgess XX45. C-1. 1/2 mf, rated voltage 1000. C-2. 0.0001 mf. R-1. 1/4 meg. R-2. 5 meg. R-3. 500 meg. T-1. Locher stratosphere Geiger-Müller tube. T-2. Radio tube type 1T4.

In the compact arrangement shown here, the coil is removed from its wooden box and insulating compound, but some trouble can be saved if a slightly larger metal box is used to house the instrument. The size of the instrument has been considerably reduced by the use of a 1T4 for rectifier and another for amplifier instead of the larger tubes. Despite their miniature size they stand up under operation and even intentional abuse.

few hours of service, vibrators may constantly change behavior, but once they become worked in, it is seldom necessary to adjust the knob. This is the only adjustment of any kind, voltage being regulated to give such a reading on the meter as previously found best for the operation. Most of the tubes operate best with a cathode potential of about minus 750 volts, but this varies somewhat for each Locher tube.

The one tube amplifier gives a sufficiently strong signal easily heard in noisy places. Though the click can be heard without any amplification if good phones are used, such an arrangement is useless in laundries, sewage disposal plants, and other places where the noise level is high.

The critical part of any Geiger-Müller counter circuit is the small coupling condenser which is shunted by the high ohmage resistor. An ordinary good grade "postage stamp" condenser is suitable, but it is recommended that the best one be picked from a lot of several.

With the batteries suggested here, the normal life is about one year with frequent use.

An instrument of this kind when kept in good condition and tested once every few weeks is a great potential safeguard to all the radium in the community.

103 Rutledge Avenue
Charleston, S. C.

REFERENCE

TAFT, ROBERT B.: Two Small Portable Geiger-Müller Counters. Review of Scientific Instruments 11: 63, February 1940.

A Rapid Dark Adaptation Test[1]

ROGER A. HARVEY, M.D.

Rochester, N. Y.

THERE ARE NUMEROUS methods for determining dark adaptation time, but most of these require complicated apparatus. The method which is to be described is not fundamentally original (1), but is simple and is particularly useful in fluoroscopy rooms for checking the state of dark adaptation of the examiner or of observers who frequently claim to be fully adapted long before such a stage has been reached.

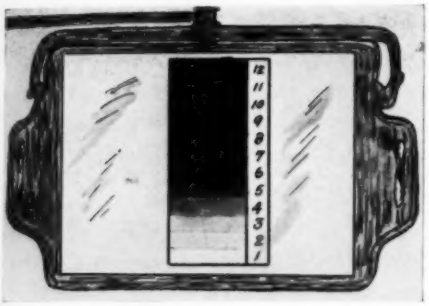


Fig. 1. Showing film against the fluoroscopic screen.

For this test an aluminum step tablet having 12 steps, each 2.5 cm. in width, 10 cm. in length, and progressively increasing 1 mm. in thickness, was placed upon x-ray film. Lead numbers from 1–12 were placed on, or between, or beside the steps, and the film was exposed to an x-ray beam. The processed film showed the following readings on a densitometer: step 1 equalled 1.7 density units, step 2 equalled 1.92, step 3 equalled 2.13, step 4 equalled 2.42, but beyond this step the density was too great to be measured on the densitometer available. Where an aluminum step tablet is not available, strips of film can be arranged in order of increasing density to simulate the one described. Films of different density scale may be required for various needs.

In measuring dark adaptation, the film is placed against the fluoroscopic screen, which is allowed to fluoresce under the film area only. The steps in the film become clearly visible—the number of steps seen depending upon the observer's degree of dark adaptation (Fig. 1). Unnecessary fluoroscopic exposure can be avoided by limiting the periods of test observation to five seconds each, or by placing the film on a rectangular-shaped box having a pale

Fig. 2. Showing film on box lighted by a fluorescent bulb.

blue glass cover and a 15-watt fluorescent electric bulb (Fig. 2).

There are definite procedures which must be followed if comparative tests are attempted with this device. Of these procedures, numbers 1, 2, and 5 do not apply to the examiner, merely checking his adaptation state prior to fluoroscopy:

(1) Explanation of the film so that the person being tested will know what to expect.
(2) A standard period of pre-exposure to a source of constant brightness.
(3) A fixed distance from eye to film.
(4) A fixed distance from x-ray tube to fluoroscopic screen.
(5) A standard period of observation at predetermined intervals.

In testing a group of 14 doctors and med-

[1] From the Department of Radiology of the University of Rochester School of Medicine and Dentistry and the Strong Memorial Hospital. Accepted for publication in November 1941.

ical students by this method, it was found that dark adaptation times varied from three to twenty minutes. The short times were in persons who were known to be taking large supplements of vitamin A. Similar variations were noted in the radiologists tested, but even more striking were the individual variations observed during a single day. Early in the morning, the adaptation time might be as low as three minutes, but later in the day it might increase to fifteen minutes in the same person. This wide range in accommodation time can be accounted for by the depletion of visual purple from prolonged exposure to intense illumination during the interpretation of routine films. This variation reveals the fallacy of any set time for dark adaptation before beginning a fluoroscopy, and demonstrates the aid which a simple test procedure can render.

Although the method described does not have the accuracy of the biophotometer and similar instruments, it has met local requirements satisfactorily, and can be used wherever an inexpensive, rapid test of dark adaptation is desired. The strip of film described requires adaptation to step 9 for chest fluoroscopy, and to step 12 for gastro-intestinal work. These figures apply to the installations used, and may vary slightly with other fluoroscopic equipment.

Strong Memorial Hospital,
Rochester, N.Y.

REFERENCE

1. CHANTRAINE AND CRAMER: Zur Prüfung des Dunkelsehvermögens. Röntgenpraxis 9: 329-336, May 1937.

CASE REPORTS

Marble Bones with Pathological Fracture and Bilateral Optic Atrophy in a Negro Child[1]

BENJAMIN W. ANTHONY, M.D., and
HERBERT C. POLLACK, M.D.
Chicago, Ill.

Marble bones, Albers-Schönberg disease, or osteopetrosis is a relatively rare condition of the human skeleton characterized by a striking change in the appearance and structure of the affected bones.

This disease may involve the entire bony framework, but has a special predilection for the long bones, being more pronounced at their ends. It is almost exclusively diagnosed roentgenologically. The involved bones appear uniformly white, dense, and structureless, with partial or complete obliteration of the medullary spaces. X-ray films of such bones, therefore, appear to have been underexposed. Additional films taken with increased exposure still show these bones to be extraordinarily opaque, and further roentgen studies of other regions of the skeleton reveal a more or less generalized pathological condition. Whereas osteopetrosis is found predominantly in early childhood, it has also been observed in both uterine and adult life. It is most frequently discovered by chance on x-ray examination of an injured extremity or during the course of a routine radiological examination. As "marble bones" are extremely brittle, minimal trauma is apt to produce a fracture—a so-called pathological one.

CASE REPORT

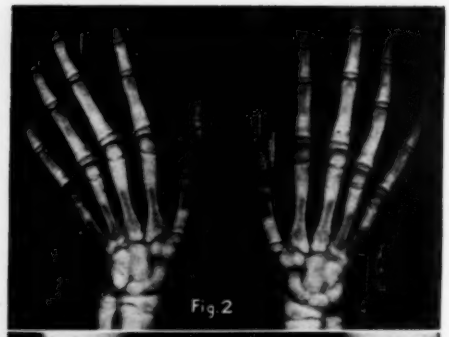
A. M. P., a 9-year-old colored girl, who had been blind for the past seven years, was admitted to the hospital on June 14, 1939, complaining of pain in the left hip and inability to walk. The pain had come on suddenly and violently as she arose from a chair. She had fallen to the floor and was unable to arise. The pain, though gradually decreasing in intensity, had continued. Her general health had been good until the present condition developed, and she was fairly well nourished.

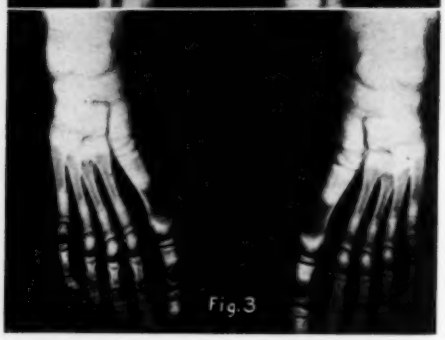
The child was unable to use her left lower extremity. The left hip was painful, swollen, and showed abnormal mobility. The left leg was 2 cm. shorter than the right. The examination was otherwise negative except for the fact that the patient was blind, as mentioned above. Tuberculosis of the left hip was suspected, with pathological fracture of the proximal end of the femur.

Fig. 1. Left hip joint and femur: incompletely healed pathological fracture, slight deformity, articular space narrowed, femur and os coxae sclerosed.

X-ray examination of the pelvis revealed a subtrochanteric fracture of the left femur with moderate overriding of the fragments (Fig. 1). It was at once evident that we were dealing with a classical case of Albers-Schönberg disease, since all of the

[1] From the Department of Roentgenology, Provident Hospital, Chicago. Chairman, Benjamin W. Anthony, M.D. Accepted for publication in November 1941.





Figs. 2 and 3. Peculiar distribution of sclerosis involving all bones of hands and feet, especially first metacarpals and metatarsals.

pelvic bones and the proximal ends of the femora showed an extraordinary degree of sclerosis. The fracture was treated in the typical way, leaving only minimal deformity. The angle between the femoral head and shaft, however, was considerably increased, with resulting coxa valga, by virtue of which a shortening of the extremity was prevented. Healing was complete within six weeks, although an abnormally small amount of callus was deposited at the site of the fracture.

A roentgenographic examination of the patient's entire skeleton was done five months after occurrence of the fracture of the left femur and practically all the bones showed findings characteristic of osteopetrosis. The bones of the hands and feet showed sclerosis of the proximal portions of the first metacarpals and metatarsals as well as the shafts of all of the phalanges (Figs. 2 and 3). The second to fifth metacarpals and metatarsals revealed sclerotic changes at their distal ends. The medullary spaces in the involved portions of the bones were obliterated. The epiphyses of these bones showed only a moderate sclerosis; the epiphyseal lines and articular spaces appeared normal. The carpal bones showed the lamellated appearance typical of this condition. The proximal three-fourths of the humeri were sclerosed and the medullary spaces were almost obliterated (Fig. 4). The distal portions of the radii and ulnae were extremely dense, particularly near the wrists.

The bones of the shoulder girdle, the ribs, the thoracolumbar vertebrae, sacrum, and pelvis were involved to a pronounced degree. An irregular, marble-like appearance was noted in all of the ribs (Fig. 5). The four boundaries of the bodies of the thoracic and lumbar vertebrae were shown to be sclerosed in the films in the frontal projection (Fig. 6). In the lateral view the sclerosis presented in the form of a "C," leaving the central portion and the anterior aspect of each vertebral body free (Fig. 7). This absence of involvement of the medullary

Fig. 4. Sclerosis and almost complete obliteration of the medullary spaces of the proximal three-fourths of the humerus. Radius and ulna involved to a less extent.

or central portions of the vertebral bodies was in contrast to the process in the long bones. The sacral and pelvic bones were sclerosed and the ilia were lamellated. The contour of each acetabulum was irregular and the joint spaces of the hips were narrowed due to abnormal bone deposits.

Each femur presented an extremely bizarre appearance in that its distal two-fifths was enormously expanded in the form of a club (Fig. 8). The widened portions of these bones presented transverse bands alternating in density, giving a zebra-like appearance. In the involved portions of the bones, there was no definite differentiation between the cortex and medullary space. Tibiae and fibulae were uniformly sclerosed at the proximal ends and striated in the lower three-fifths (Fig. 9). The stripes of negative density probably represented

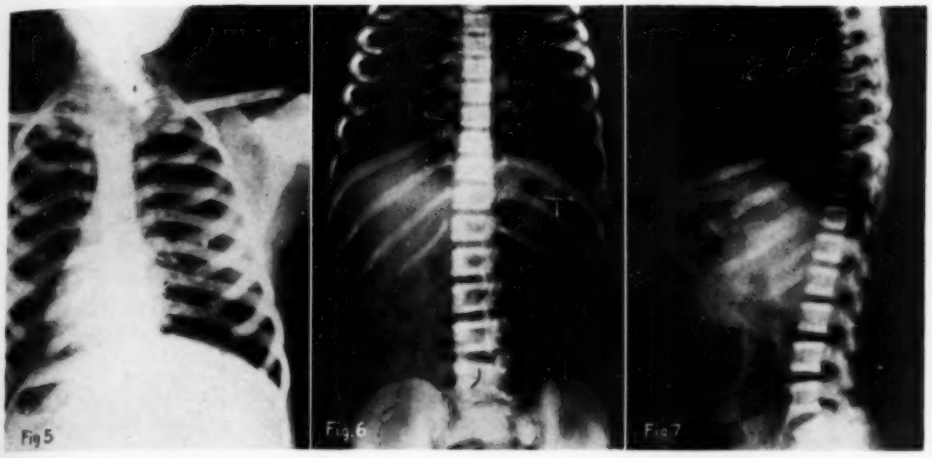


Fig. 5. Marble-like appearance of ribs, clavicles, and right scapula.
Fig. 6. Thoraco-lumbar spine and ilia, frontal projection, showing the pathological changes in the vertebrae and concentric lamellation of the iliac bones.
Fig. 7. Thoraco-lumbar spine, lateral view. The cortical margins of all the vertebral bodies are markedly sclerosed except for the anterior aspect.

areas of decreased resistance and therefore possible sites of pathological fractures.

Special attention was paid to the skull, which was extraordinarily thick, with sclerosis of the bones of its base, best demonstrated in the lateral view (Fig. 10). From these findings one would expect all apertures at the base of the skull to be encroached upon. Separate films were therefore made of the optic foramina, which were found to be uniformly narrowed to a pronounced degree, the diameter being but 2.4 × 3 mm. The average normal measurements are 4.1 × 4.65 mm. according to Clegg, who found no variation due to age, sex, or stock and concluded that an optic foramen measuring less than 2.8 mm. in any one of its diameters cannot contain a normal optic nerve.

Figure 11 permits a comparison of a normal optic foramen and that of our patient showing concentric constriction, which obviously represents a part of the proliferating bony process at the base of the skull. The gradually increasing stenosis of the optic foramina explains the slowly progressive blindness in this case. According to the history, the child was able to see normally until she was two years of age, at which time a slowly advancing loss of vision occurred, leading to complete blindness within three months. The pupils were equal, fixed, and did not react to light or accommodation. Slight opacity of the lenses and nystagmus were noted. There was complete bilateral optic atrophy.

Published records of the blood findings in this disease show a decided tendency toward anemia. The blood chemistry has been reported as normal by all observers except Kopylow, who found a hypercalcemia in his patient.

In our case the white cell count was 7,900, with a normal differential count; red cell count 3,900,000; hemoglobin 73 per cent. Blood chemistry showed non-protein nitrogen 37.9 mg. per cent; glucose 69 mg. per cent; cholesterol 148.3 mg. per cent; serum calcium 10.8 mg. per cent; serum phosphate 5.93 mg. per cent.

The decreased number of red cells (3,900,000) and the lowered hemoglobin (73 per cent) indicate a moderate anemia. These findings are compatible with the diminished medullary spaces, which constitute a considerable portion of the hemopoietic tissue. There was no disturbance in dentition and the condition of the teeth was good. The physical development of the patient, however, corresponded to that of a child two years younger, confirming the opinion that, if the disease is manifested in early youth, a disturbance in growth is to be expected. There was nothing in the history of our patient to indicate that this condition was either of hereditary or familial origin.

A review of the literature reveals about 118 cases of osteopetrosis, a malady which was first described in 1904 by Albers-Schönberg, after whom the condition is named. The most recently reported case is that of Vidgoff and Bracher in 1940. Despite the fact that a great number of reports concerning osteopetrosis have appeared, its etiology is still unknown. The most generally accepted view is that it is due to a disturbance in calcium metabo-

lism, but its exciting cause has not been discovered.

Péhu found in his case of marble bones a tumor of the parathyroid gland and was of the opinion that this might be the underlying causative factor. With the exception of Kopylow, however, no one has demonstrated abnormal amounts of calcium in the blood. Pounders and Pirie believe the condition to be a familial, hereditary, and constitutional disease which may begin *in utero*. Pirie was inclined to regard consanguinity, infection, and diet as responsible factors. Istria believes the condition to be due to an endocrine disturbance, *e.g.*, of the parathyroid, thyroid, thymus, or hypophysis. Wortis, on the other hand, assumed that osteopetrosis might be attributable to a developmental defect, as he found this disease in patients with undescended testes, club foot, etc.

Albers-Schönberg disease, as a rule, is more or less symptomless, usually being found by chance during a roentgen examination done for other reasons. Often, however, one or several of the following signs and symptoms may be present: pathological fractures; chronic osteomyelitis; deformities of the skeleton, especially of the epiphyseal areas; physical underdevelopment; anemia; enlargement of the liver, spleen, and lymph nodes; optic atrophy with blindness or diminution of vision; hydrocephalus with mental retardation; delay in walking and dentition; defective teeth together with alveolar necrosis.

In almost all of the cases pathological fractures, either single or multiple, have led to the diagnosis of marble bones. These fractures are painless, usually transverse, not splintered, and are accompanied by very little swelling. They involve the ends of the long bones almost exclusively and occur after the slightest trauma.

For the most part there is an abnormal proliferation of amorphous opaque tissue beneath the periosteum and cancellous tissue from the endosteum, producing a gradual obliteration of the medullary canal. The periosteum, however, does not usually

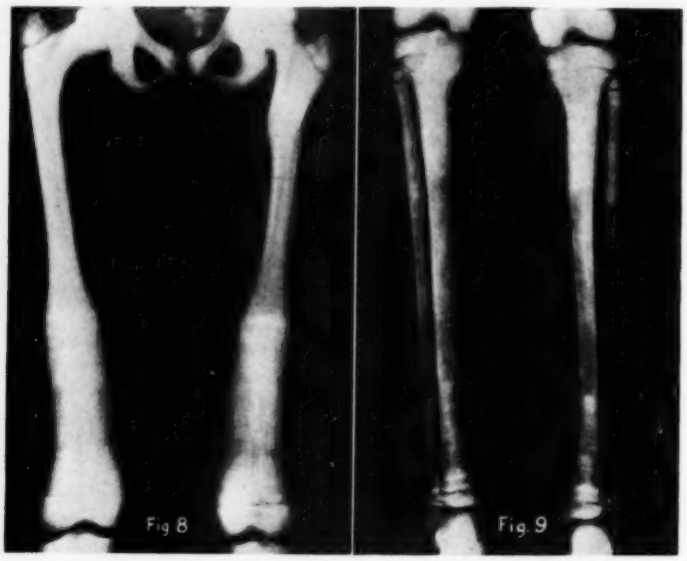


Fig. 8. Ischia and pubes as well as proximal ends of femora are densely sclerosed. Club-shaped distal ends of femora with zebra-like formations.
Fig. 9. Tibiae and fibulae uniformly sclerosed at the proximal ends and striated in the lower three-fifths.

participate in the bony changes. The involved bones, though they appear extremely firm and almost unbreakable, are in reality very brittle and break easily due to loss of elasticity. Pirie found that pieces of osteopetrotic bones were more easily penetrated by a drill than normal ones and for this reason he recommended the term "chalky bones" as a better expression than "marble bones." Fractures of such bones heal at a relatively normal rate.

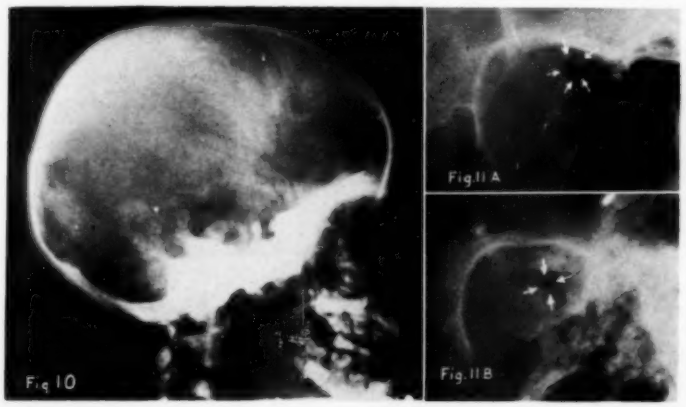


Fig. 10. Lateral view of skull, showing pronounced thickening and sclerosis of the entire base.

Fig. 11. A is a normal optic foramen, 4.2 × 4.8 mm. in diameter, of a girl of the same age as the patient. B is the pathologically constricted optic foramen, 2.4 × 3 mm. in diameter, with total optic atrophy.

SUMMARY

A case of generalized marble bones, exhibiting a pathological fracture of the femur, has been described. The patient was completely blind as a result of bilateral optic atrophy secondary to constriction of the optic foramina resulting from bony proliferation at the base of the skull. Besides this, a moderate anemia and a slight diminution in growth were the outstanding pathological findings. The blood chemistry in such cases has been found normal.

426 East 51st St., Chicago, Ill.

REFERENCES

McCUNE, DONOVAN J., AND BRADLEY, CHARLES: Osteopetrosis (Marble Bones) in an Infant: Review of Literature and Report of Case. Am. J. Dis. Child. **48:** 949–1000, November 1934.

DAVIS, GEORGE G.: Osteosclerosis fragilis generalisata. Arch. Surg. **5:** 449–463, November 1922.

Editorial: Albers-Schönberg's Disease. Am. J. Roentgenol. **33:** 94–96, January 1935.

GOLDEN, Ross: Diagnostic Roentgenology. New York, Thos. Nelson & Sons, pp. 369–371.

D'ISTRIA, A.: A Case of Marble Bones. Radiol. med. **15:** 473–485, May 1928.

JENKINSON, E. L., AND LEWIN, PHILIP: Bone Diseases in Infancy and Childhood. Am. J. Roentgenol. **17:** 201–214, February 1927.

KERR, H. DABNEY: Case of Osteopetrosis (Marble Bones), Complicated by Osteogenic Sarcoma. Am. J. Roentgenol. **35:** 212–214, February 1936.

KOPYLOW, M. B., AND RUNOWA, M. F.: Contribution to the Knowledge of Marble Bones. Fortschr. a. d. Geb. d. Röntgenstrahlen **40:** 1042–1054, December 1929.

KRAUS, E. J., AND ARNO, W.: Albers-Schönberg's Disease. Med. Klinik **21:** 19–22, Jan. 4, 1925.

MERRILL, A. S.: Case of Marble Bones with Pathological Fractures. Am. J. Roentgenol. **21:** 361–362, April 1929.

MOREAU, L.: Roentgen Considerations on Certain Osseous Affections. Bull. et mém. Soc. radiol. méd. de France **19:** 471–477, November 1931. Abst. in Am. J. Roentgenol. **29:** 555, April 1933.

PÉHU, M., POLICARD, A., AND DUFOURT, ANDRÉ: Osteopetrosis or Marble Bones: Presse méd. **39:** 999–1003, July 4, 1931. Abst. in Am. J. Roentgenol. **29:** 563, April 1933.

PIRIE, A. HOWARD: Development of Marble Bones. Am. J. Roentgenol. **24:** 147–153, August 1930.

PIRIE, A. HOWARD: Marble Bones. Am. J. Roentgenol. **30:** 618–620, November 1933.

POUNDERS, CARROLL M.: Congenital Osteosclerosis. Ann. Int. Med. **8:** 966–971, February 1935.

ROOT, J. HAROLD: Albers-Schönberg's Disease. Am. J. Dis. Child. **49:** 964–973, April 1935.

STENSTRÖM, B.: Marble Bones. Acta Radiol. **17:** 140–151, 1936.

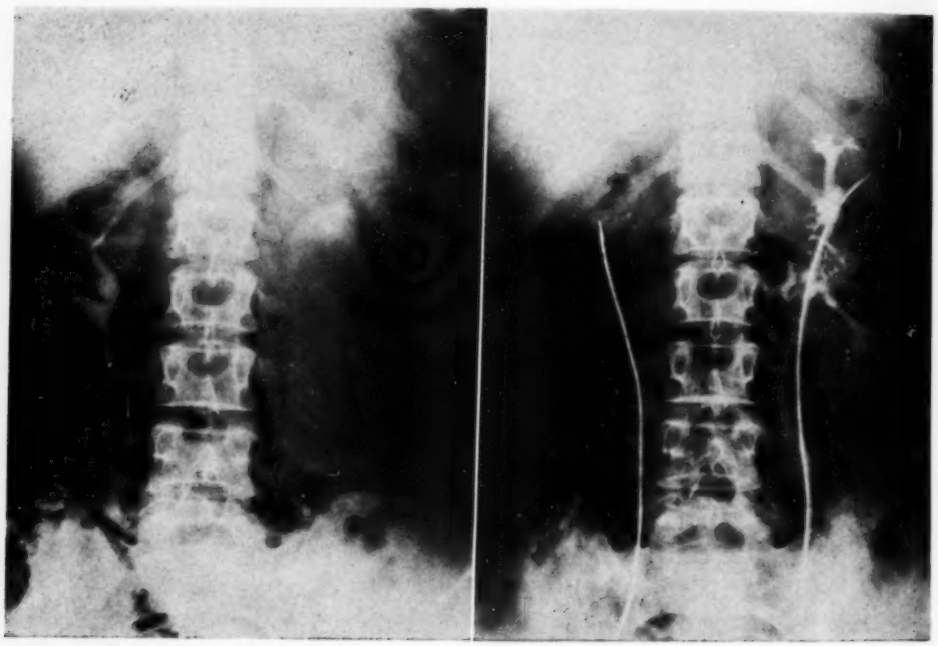
VIDGOFF, BEN, AND BRACHER, GEORGE J.: Osteopetrosis. Am. J. Roentgenol. **44:** 197–202, August 1940.

WORTIS, HERMAN: Osteopetrosis (Marble Bones). Am. J. Dis. Child. **52:** 1148–1157, November 1936.

Rupture of the Kidney Diagnosed by Retrograde Pyelography[1]

ALEXANDER J. CHILKO, M.D.
New Rochelle, N. Y.

Traumatic rupture of the kidney is an uncommon but not rare event. Its occurrence is estimated at one to every 1,000 to 3,000 injuries. The clinical picture is fairly characteristic, the main features being pain, tumor, hematuria, ecchymoses, shock. If a history of trauma is given, the diagnosis is usually not difficult. If, on the other hand, nothing is known about the trauma it is hard enough to recognize the condition. This occurred in a case recently observed.



Figs. 1 and 2. Rupture of left kidney: intravenous pyelogram (left) and retrograde pyelogram (right).

CASE REPORT

Mrs. E. F., a 51-year-old white woman, complained of acute, sharp, cutting pain radiating down the left thigh, accompanied by blood in the urine, nausea, and vomiting. Absolute bed rest failed to stop the bleeding and her physician referred her to the New Rochelle Hospital for further observation. The possibility of a renal tumor was considered most likely. Five days after admission the condition of the patient permitted cystoscopic examination. The bladder was found to be perfectly normal. Pure blood was seen coming from the left ureter. Indigo carmine injected intravenously appeared on the right side in 5 minutes and on the left in 10 minutes.

A flat roentgenogram of the abdomen showed the shadow of the left kidney to be immensely enlarged, reaching from the 11th dorsal to the 4th lumbar vertebra. There was no evidence of any radiopaque lithiasis which could have explained the bleeding. Intravenous pyelography tended to confirm the suspicion of a tumor of the left kidney. Only the upper calyx was filled by the contrast substance. A diffuse accumulation of dye was visible in the region of the upper part of the pelvis. The middle and lower parts of the pelvis and calyces were not filled at any time (Fig. 1).

A retrograde pyelogram showed all the calyces to be well filled by the contrast substance, not widened, and not deflected from their normal place in the kidney. There was a fairly large distance between the upper calyx on the one hand and the middle and lower calyces on the other. This space.

[1] From the Department of Radiology in the New Rochelle Hospital. Accepted for publication in August 1941.

which should have been occupied by the kidney pelvis, showed only patchy accumulations of dye with very irregular outlines. The tip of the catheter was found to be outside of the calyces (Fig. 2).

With these findings at hand, the idea of a neoplasm had to be abandoned. Enlargement of the kidney by a parenchymal tumor would have caused deflection of one or more of the calyces, while a hydronephrosis or pyonephrosis would have caused enlargement of the calyces. The presence of a rupture of the kidney was therefore suspected and on further questioning was confirmed, the patient admitting that she had slipped and fallen on the edge of the bath tub, striking her left flank.

Fig. 3. Ruptured kidney, surgical specimen.

In view of the continuous bleeding, a nephrectomy was done. It was found that the left kidney, together with its capsule, was torn entirely in two parts, at the level of the hilum. These two parts were kept together only by a few fibrous bands and by the wall of the pelvis. There was a gap of 2 1/2 inches at the periphery between the upper and lower half of the organ (Fig. 3). The renal artery and vein were completely severed from the kidney and blocked by thrombosis, sealing them completely, so that not a drop of blood escaped during the operation. An old organizing hemorrhage was found surrounding the kidney.

All these findings would indicate that the rupture was small at first and progressed slowly throughout the entire width of the kidney.

A month after the operation, the patient was discharged from the hospital. At present she is in perfect health.

650 Main St., New Rochelle, N. Y.

Cavum Septi Pellucidi Biloculare[1]

M. H. POPPEL, M.D.

Roentgenologist, Kings County Hospital, New York City

The purpose of this paper is to record a case in which the cavity of the septum pellucidum was biloculated as the result of a vertical mid-line adventitious partition.

The normal septum pellucidum is a thin vertical partition composed of two thin laminae in apposition with each other in the median plane and separated by a narrow median cleft of variable size which is known as the cavum septi pellucidi.

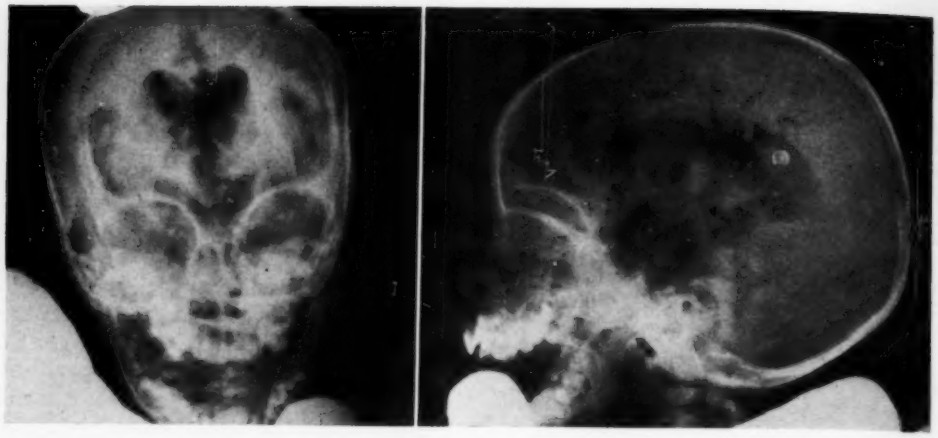
The septum pellucidum is triangular in shape and intervenes between the two lateral ventricles. Posteriorly it is prolonged backward for a variable distance between the body of the corpus callosum and the fornix, to both of which it is attached. In front it occupies the gap behind the genu of the corpus callosum, and below it is prolonged downward in the paraterminal body toward the base of the brain.

On the normal encephalogram the septum pellucidum is represented by a well demarcated vertical linear shadow, about 2 or 3 mm. in width, interposed between the air shadows of the lateral ventricles in the anterior-posterior or posterior-anterior view.

Abnormally, more often in infants, the two leaflets are separated and form the lateral walls of the cavity of the septum pellucidum. This cavity, known also as the fifth ventricle, may be subdivided by the apposition of the fornix and the corpus callosum into an anterior portion, for which the designation fifth ventricle is retained, and a posterior portion or sixth ventricle.

Congenital absence of the septum pellu-

[1] From the Department of Radiology of the Kings County Hospital of the Department of Hospitals of the City of New York, Dr. Richard A. Rendich, General Director. Accepted for publication in January 1940.



Figs. 1 and 2. Cavum septi pellucidi biloculare: anterior and lateral views.

cidum alone is very rare. Perforation of the septum is usually associated with internal hydrocephalus, of which a considerable number of cases have been reported. More often, absence or defects of the septum are accompanied by defects in the corpus callosum and other variations in the brain. Secondary septal perforations may occur in cases of tumor of the third ventricle with obstruction of the foramen of Monro and internal hydrocephalus.

No example of biloculation of the cavum septi pellucidi in a vertical direction in the mid-line could be found in the literature.

CASE REPORT

A one-year-old colored girl was admitted because of general underdevelopment. Her weight at birth, at full term, was 4 pounds; at one year it was 12 pounds.

The child was the first born to a seventeen-year-old mother, whose previous and present history was normal. Examination of the patient revealed underdevelopment both physically and mentally. The Wassermann test was negative. The blood count and chemistry were normal.

Stereoscopic roentgen examination after encephalography revealed a cavity of the septum pellucidum which was biloculated by a vertical midline partition. This was associated with a slight generalized internal hydrocephalus and generalized cerebral agenesis.

114 E. 54th St., New York, N. Y.

REFERENCES

CUNNINGHAM, D. J.: Textbook of Anatomy, New York, Wm. Wood & Co., 5th Edition, 1923.

DYKE, C. G., AND DAVIDOFF, L. M.: Congenital Absence of the Septum Pellucidum. Am. J. Roentgenol. **34:** 573–578, November 1935.

Prolapse of Polypoid Gastric Mucosa into the Duodenum, with Malignant Change[1]

JACOB S. RUBIN, M.D.
Jamaica, N. Y.

A review of the literature as far back as 1923 reveals little concerning the prolapse of malignant growths of the gastric mucosa into the duodenum. Many reports of benign gastric mucosal polyps have appeared but in relatively few instances has there been found actual prolapse of the tissue into the duodenal bulb, and in even fewer cases have malignant changes been observed. The most comprehensive reviews of pedunculated tumors with prolapse through the pylorus are those of Eliason, Pendergrass, and Wright in 1926, and by Pendergrass in 1930. The general conclusions drawn by these writers may be summarized as follows:

Complaints: The patient may complain of epigastric pain, paroxysmal in character and radiating to the back; also

[1] Accepted for publication in November 1941.

Fig. 1. Prolapse of gastric mucosa: six-hour roentgenogram, erect position. Note gastroduodenal residue and irregular motility.

Fig. 2. Prolapse of gastric mucosa: roentgenogram showing filled stomach, in prone position.

of oral bleeding or melena. The pain appears to have no definite relation to meals. Vomiting is not common.

Physical and Roentgen Findings: The condition is rarely conclusively diagnosed clinically. Roentgen findings are characteristic. There is a central filling defect in the duodenal bulb, with some streaking of the contrast media about it. The defect may not appear with the patient erect but is demonstrable in the prone or right oblique posture. There is no disturbance in peristalsis unless the tumor has undergone malignant change with infiltration of the muscular coat of the stomach. A varying degree of six-hour residue is observed depending upon the degree of ball-valve action. No gastric defect is visible with prolapse of a pedunculated tumor through the pyloric ring. With prolapse of gastric mucosa the appearance may resemble the deformity observed when there is slight pressure of the pyloric region on the spine.

Anemia of the secondary type is present.

Pathology: Papillomas, adenomas, fibromas, polyps, multiple or single, are usually found in the stomach near the pyloric ring. These are prone to ulcerate, with resulting bleeding and anemia. The origin is usually a low-grade inflammation from a chronic irritation, with eventual mucosal hypertrophy, and an attempt to expel the mass by peristalsis, as in the presence of a foreign body.

Differential Diagnosis: Differentiation from ulcer is possible only by x-ray studies. The secondary anemia due to bleeding is to be distinguished from pernicious anemia.

CASE REPORT

J. T., a white female aged 63, was first seen by Dr. G. March in 1940, complaining of heartburn, nausea, epigastric distress with all foods, and a loss of weight amounting to 7 pounds. These symptoms were of four months' duration. Tarry stools had been observed for one month.

The early history and family history were irrelevant. Epigastric distress at first occurred only upon ingestion of solid foods, but later with liquids as well. Relief was occasionally obtained by vomiting. Otherwise the pain lasted for hours.

Physical examination showed general arteriosclerosis, soft systolic murmurs in the mitral and aortic regions, and slight cardiac enlargement. Distended, tortuous veins were present on the thin-walled abdomen. The blood pressure was 120/76. A small lump, 2 or 3 cm. in diameter, was felt through the abdominal wall in the pyloric region. Rectal examination was negative; vaginal examination was not permitted.

X-ray studies having been refused by the patient, she was placed on a dietetic régime with vitamins, and antispasmodics. She was not seen again until fifteen months later, in June 1941. She had grown steadily worse, had lost 20 pounds, and was unable to retain even liquids. The abdominal epigastric mass remained unchanged in size, but pain now radiated to the back, and the patient complained of severe constipation. A tentative diagnosis of a malignant tumor was made.

The findings on roentgen examination were as follows. The chest was essentially negative. A small six-hour gastroduodenal residue was present. The stomach showed moderate hypersecretion, slight pyloric narrowing, and rapid emptying, with active peristalsis. The bulb was large and tender. At all times it had a "cottony" appearance and sluggish motility. At twenty-four hours there was some delay in colonic motility; tenderness and limited mobility of the ileocecal area were observed. At forty-eight hours, the meal had reached only to the sigmoid colon. Tenderness was still present in the ileocecal region.

The *diagnosis* was (1) polypoid herniation of gastric antral mucosa into the duodenal bulb; (2) chronic low-grade ileocecal inflammation.

The patient was hospitalized on July 5, 1941. The blood count was: red cells 3,250,000; white cells 7,200, with a normal differential count. The urine was negative.

Operation was done on July 11. The liver showed signs of hepatitis. The gallbladder and cystic duct contained several calculi, and some adhesions were present. The right ovary was enlarged and contained a hard mass, believed to be a Krukenberg tumor. The stomach was normal on the surface, but a mass was felt in the pyloric region, which could be pushed freely into the duodenum. The lower 7 cm. of the stomach and 1 cm. of the duodenum were removed and a gastrojejunostomy was performed. No interference in the right ovary or gallbladder was deemed advisable, due to the patient's condition. The pathologist's report was as follows:

"The specimen consists of a segment of stomach measuring 7 cm. in length. On the inner surface are found one large polypoid mass, about the size and shape of a plum, and numerous small polyps of varying size. The serosal surface is fairly smooth. On section, the mucous lining appears to be intact throughout, with no gross evidence of extension into the stomach musculature.

"Microscopic sections through the polypoid mass show a thin, highly vascular connective-tissue stem covered by a hyperplastic proliferating glandular epithelium. The cells show marked hyperchromatic changes, and occasional mitotic figures are present. There is a distinct invasion of the stroma supporting these papillary folds. The sides of the connective-tissue stem are lined by normal gastric mucosa, in striking contrast to the cells composing the polypoid mass. The transition from one to the other is abrupt.

"*Diagnosis:* Gastric polyposis with carcinomatous degeneration."

The patient is still alive and relatively well at the date of writing, nearly three months after the operation.

I am indebted to Dr. Michael Gosis, the surgeon, for a very thorough case report.

90-26 148th St., Jamaica, N. Y.

BIBLIOGRAPHY

PLATT, O. R., WALTEMATH, G. F., AND MILLHOUSE, J. H.: Exogastric Pedunculated Myosarcoma of Stomach. Nebraska M. J. **25:** 336–337, September 1940.

MCLAUGHLIN, C. W., AND CONLIN, F.: Pedunculated Gastric Tumors. Am. J. Surg. **46:** 250–258, November 1939.

PENDERGRASS, E. P.: Prolapsed Pedunculated Tumors of Gastric Mucosa through Pylorus into Duodenum: Roentgenologic Diagnoses. J. A. M. A. **94:** 317–321, Feb. 1, 1930.

RIGLER, L. G.: Roentgen Observations of Stomach. Benign Tumor Prolapsing through Pylorus. Am. J. Roentgenol. **20:** 529–531, December 1928.

MILLAR, T. M.: Pedunculated Extragastric Leiomyoma of Stomach. Brit. J. Surg. **16:** 323–326, October 1928.

ELIASON, E. L., PENDERGRASS, E. P., AND WRIGHT, V. W. M.: Roentgen-Ray Diagnosis of Pedunculated Growths and Gastric Mucosa Prolapsing through Pylorus. Am. J. Roentgenol. **15:** 295–322, April 1926.

MATAS, R.: Pediculated Polypoid Fibro-Adenoma of Stomach of the Ball-Valve Type, Causing Unusual and Complex Syndrome. Surg., Gynec. & Obst. **37:** 723–731, December 1923.

Localized Aseptic Necrosis and Rarefaction of Bone

Without Obvious Fracture and Involving the Shaft of the Proximal Phalanx of the Right Index Finger[1]

THOMAS HORWITZ, M.D.
Philadelphia, Pa.

The osseous lesion to be reported here represents an unusual clinical and roentgenographic expression of aseptic necrosis and postnecrotic rarefaction of bone.

[1] Accepted for publication in September 1941.

This diagnosis was not considered preoperatively, and was established only after histologic examination. The removal of the localized disease process, under roentgenographic control, was followed by immediate and thus far continued recovery from the painful disability of one year's duration.

CASE REPORT

History: A 33-year-old steel-mill worker complained of painful swelling on the ulnar aspect of the proximal phalanx of the right index finger. There was no definite history of trauma, but the patient stated that his work required repeated and firm gripping of the handle of a machine with his right hand. At the onset, one year previously, there had been only sensitiveness to pressure, but four months later the patient began to experience throbbing, nocturnal pain at the base of the right index finger. Following a direct minor blow six months before examination, he began to have more severe and constant pain, with increased sensitiveness in the proximal portion of the finger, and local swelling appeared on the ulnar aspect.

Physical examination revealed swelling, local heat, and exquisite tenderness over the ulnar aspect of the proximal phalanx of the right index finger. The skin in this region was not discolored. The remaining portions of the finger were uninvolved, and motion in the metacarpophalangeal and interphalangeal articulations was unimpaired. Examination of the remainder of the musculo-skeletal system and of the other systems of the body was negative.

Roentgen examination of the bones of the right index finger showed a small circular radiolucent area on the ulnar surface of the distal end of the shaft of the proximal phalanx. The thin overlying cortical bone was unbroken, but was elevated like a blister. The bone immediately adjacent to this rarefied bony defect was less dense than the bone of the remainder of the proximal phalanx, of the other phalanges, and of the other hand bones, all of which appeared normal. The ulnar border of the phalanx, for about one centimeter proximal to the circular rarefied defect, appeared irregular, and at one point a definite groove was demonstrable. The soft tissues overlying these bony changes were thickened (Fig. 1). A roentgenogram of the left hand revealed no abnormalities.

The blood picture was a normal one. The blood Wassermann and Kahn tests were negative. An intracutaneous tuberculin skin test was negative. Repeated urinalyses revealed normal findings.

Operation (Nov. 27, 1940): Through an incision on the dorso-ulnar aspect of the proximal phalanx of the right index finger, the small area of involved bone was completely excised, under roentgenographic control. The proximal interphalangeal articulation was not disturbed. Although the soft tissues were thickened and somewhat congested, there was no gross evidence of a specific pathologic lesion. The surface of the bone, prior to excision, and the denuded bone, following the ostectomy, appeared grossly normal.

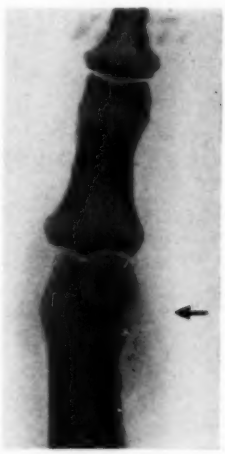


Fig. 1. Postero-anterior roentgenogram of the phalanges of the right index finger. There is a small circular radiolucent defect on the ulnar border of the distal end of the proximal phalangeal shaft. The thin overlying cortex is unbroken and is raised like a blister. The bone immediately adjacent to this area is somewhat decalcified. The ulnar border of the phalanx, for about one centimeter proximal to the rarefied defect, is irregular, and at one point a definite groove is demonstrable.

Postoperative Course: The patient experienced immediate cessation of pain, and the pre-existing sensitiveness and tenderness were no longer present. The sutures were removed from the healed wound in one week, and motion was restored to the finger joints by intensive physiotherapy. The complete recovery has continued up to the present. Postoperative roentgenograms revealed no bony abnormalities other than a smooth defect at the operative site.

Histopathologic Findings: The area represented by the rarefied circular defect on the roentgenogram consisted largely of osteogenic connective tissue, containing occasional distended vascular spaces; considerable bone rarefaction was observed. There

were scattered spicules of living and non-viable lamellar bone, undergoing active vascular resorption. The fibrous tissue filling the interstices between or replacing the thinned bony trabeculae was quite cellular, and this was especially marked at the periphery of the spicules of partially or completely necrotic lamellar bone, where there was evidence of active osteogenesis. There was no evidence of inflammatory cell reaction (Fig. 2). Culture of some of the tissue removed from the wound revealed no bacterial growth.

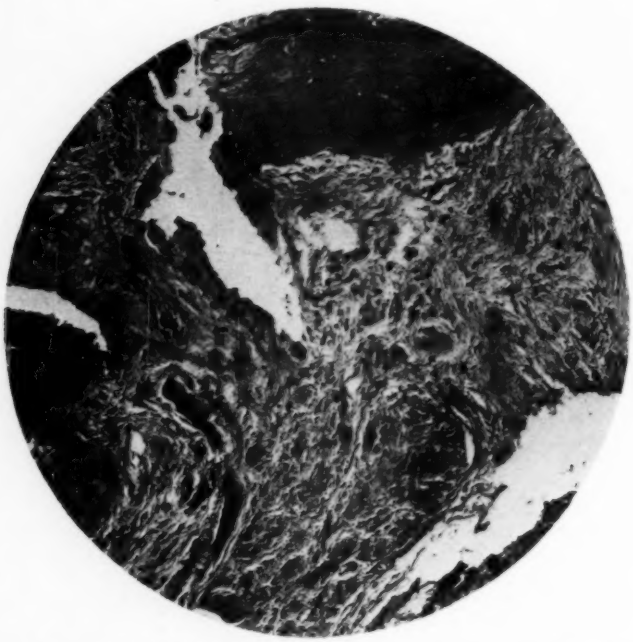


Fig. 2. Histologic section (×100) from the area represented by the rarefied circular defect in Fig. 1. There are scattered spicules of living and dead lamellar bone, which are undergoing active vascular resorption. The connective tissue filling the interstices between or replacing the bony trabeculae is quite cellular, especially at the margins of the spicules of lamellar bone, where there is evidence of active osteogenesis, with the deposition of osteoid tissue and immature bone. There is no evidence of inflammatory cell reaction.

Diagnosis: Localized aseptic necrosis and postnecrotic rarefaction of bone.

DISCUSSION

The clinical and roentgen findings were not considered to be consistent with a diagnosis of the localized infectious or benign or malignant neoplastic processes known to affect the long bones of the hand, primarily or secondarily. The diagnosis of aseptic necrosis and postnecrotic rarefaction was arrived at only after histologic examination of the material removed at operation. Although this is an unusual expression of this disease process, it is noteworthy that King has illustrated an identical localized lesion, that was productive of pain and tenderness, in the ulnar styloid process, and that he has recorded a similar observation in the head of a metacarpal bone. He has interpreted this lesion as being probably traumatic in origin. Although there is no roentgenologic or histologic evidence of an associated fracture, recent or healed, in the case here reported, the necrosis may well have followed a localized vascular lesion, with or without bony infraction. Despite the absence of a history of specific trauma, the potentialities of minor injury during the patient's laborious duties cannot be overlooked.

The histopathologic features of the osseous lesion in this case are identical with those observed in other localized rarefying diseases of bone (King). The roentgenographic picture of these lesions is dependent directly upon their histopathologic features. Those areas in which the bony trabeculae are thinned and widely separated, and the interstices are filled with vascular fibrous tissue, will appear radiolucent. The trabeculae may become compressed and roentgenographically the bone will appear densified. Rarefied areas on the roentgenogram may represent zones of widely dissimilar histologic structure—fibrous, fatty, cartilaginous, or osteoid tissues, or areas of mucoid degeneration. The radiographic appearance will be further modified dependent on the amount of bone necrosis and the degree of replacement of the necrotic bone trabeculae by new bone formation.

CONCLUSIONS

The osseous lesion described in this presentation represents an atypical expression of localized aseptic necrosis and postnecrotic rarefaction involving a single adult long bone of the hand. Immediate and thus far continued recovery from an associated painful disability of one year's duration followed complete excision of the involved bone, under roentgen control.

Physicians' Bldg.
20th and Chestnut Sts., Philadelphia, Pa.

REFERENCE

1. KING, E. S. J.: Localized Rarefying Conditions of Bone as Exemplified by Legg-Perthes' Disease, Osgood-Schlatter's Disease, Kümmell's Disease and Related Conditions. Baltimore, William Wood & Co., 1935.

EDITORIAL

The Beginnings of Disease

Presidential Address[1]

I am fully aware of the excellence of this occasion. I even more appreciate the small part I have had in its formation. Such an event as this, the 27th Annual Banquet of the Radiological Society of North America, is not the consummation of the labors of one man but of many men, working many hours a day for many years—a quarter of a century of united effort. There have been those far-seeing scientists, exemplified by such personages as the Orndoffs, the Soilands, and the Stevens, the Carmans, the Pfahlers, and the Ernsts, who have realized that it is not sufficient to merely have a national society but rather that such an organization has a responsibility to its members and to the people, and who have given of their very life blood to perfect this ideal. To those men with such a vision and to the leaders of today with the willingness and resolution to carry on, to the Radiological Society and its members, I extend my greetings.

Our world about us is a wonderful but a fearful world. Among its myriad activities there is a group of silent forces known as natural processes or as Nature. These processes, this system of activities, are both wonderful to contemplate and fearful to know. They create only to destroy. Atomic particles are put together to create physical bodies and taken apart to destroy them. In other words, Nature has both creative and destructive forces, and immediately upon the creation of any physical body destructive forces are set in motion which will eventually destroy it. Nothing seems to escape this ultimate end unless it is the component parts of the atoms, the matter-energy particles. The trees, the plants, the earth, the planets, must all meet the same eventual end. This state of things, which has the power to give both joy and happiness, illness and death, has produced the physician.

Into this environment of natural processes is placed the human being. We usually assume that the infant or young child is a physiological unit with properly balanced physiologic activities for growth and development. This is not the case. The infant and young child have no more an ideal physiologic state for growth and development than the mature adult has for continual life. The child of today is the adult of tomorrow. The physical qualities of the child are known as its anatomy and the changes taking place from infancy to adult life are the progression of this anatomy. Seldom, if ever, are the activities of the anatomical structures in such a state of equilibrium that they are neither progressing or receding. There is never a condition of complete rest. Even in the genes of the ovum and spermatozoa the forces of nature are already beginning to influence the future life of the human being. Upon the formation of the anatomy of the fetus, upon the birth of the infant, inherited qualities and environment begin to disturb the natural physiologic processes and to establish the first beginnings of disease. The progression of these beginnings of disease throughout the life span of the individual and the changes registered by them upon the anatomy of the individual constitute the life cycle, the story of the chronic disease which

[1] Delivered before the Radiological Society of North America at the Twenty-seventh Annual Meeting, San Francisco, Calif., Dec. 1–5, 1941.

kills at fifty. Since these chronic diseases are progressive changes in anatomy, then their detection and portrayal should be the goal of the physician and particularly the radiologist—the writing of the story of chronic disease from its beginning until death. Such a story of romance and tragedy has never been written and only partially attempted.

The physician, generally speaking, has the available time to diagnose and to treat only the acute illnesses and those end-results of many years of obscure but chronic disease. The acute illnesses are those catastrophies of life which demand immediate attention. The end-results, as gallstones or cancer, follow years of unrecognized chronic disease, Nature's processes for destruction. When the surgeon removes gallstones, he removes the end-results of disease and by so doing contributes very little to our knowledge of the beginning of the gallstones. On the other hand, what contributions to our knowledge could be had by a careful study of this gallbladder throughout the patient's life span! What scientific data might be obtained by the roentgen examination of the gastro-intestinal tract throughout the life span preceding ulcer and cancer of the stomach, or even preceding appendicitis! What radiologist but would like to have the life record of the osseous system of the patient with Paget's disease!

It is possible for the physician to observe and to record the complete life cycle of an acute illness, which may be only of a few weeks' or months' duration, but the duration of a chronic illness which kills at fifty may be fifty years. A complete study and recording of such a chronic disease can then only be made by the combined, organized efforts of many scientists. Chronic diseases are disturbances of the physiologic state and register themselves as pathologic changes of the living anatomy. Such disturbances of physiology and the pathologic changes of the anatomy can usually be demonstrated by the roentgen examination. Of all the physicians of today the radiologist has the greatest opportunity to record and to analyze the life span of the great group of chronic diseases. The roentgen examination is the best single method for portraying the functions and dysfunctions of living anatomy. Will the radiologists assume this responsibility—a responsibility of leadership? In the meantime the child, the young adult, proceeds into adult life with anatomical changes—the beginnings of serious disease—as the result of the forces of heredity, environment, and nature, unrecognized and untreated.

As "sleep" is the "chief nourisher in life's feast" so is leisure time the chief nourisher of research. As radiologists we have been occupied with developing methods for the diagnosis and the treatment of the acute illnesses and the end-results of chronic disease. This task has been wonderfully well done. May we not be approaching an opportune time for the organized study of the beginnings of disease? Such studies would not only enhance the pleasures of living but also add another decade to life's expectancy. May America continue to be blessed and may the radiologist continue to have the blessings of adequate equipment and the leisure time to further his investigations of the diagnosis and treatment of disease and especially *the beginnings of disease.*

W. WALTER WASSON, M.D.

INSTITUTE ON CANCER

A one-day Institute on Cancer was held on Jan. 17 at Syracuse University College of Medicine under the auspices of the College, the Tumor Clinic Association of the State of New York, Medical Society of the State of New York, and the Division of Cancer Control of the New York State Department of Health. The institute was a part of the program of postgraduate medical education conducted by the Medical Society.

The following program was presented:

1:30 P.M.

The Rôle of Tumor Clinics in Cancer Control, BOWMAN S. CROWELL, M.D., Associate Director, American College of Surgeons, Chicago, Illinois

Discussion: Louis C. Kress, M.D., Director, Division of Cancer Control, State Department of Health, Albany, New York

The General Practitioner and the Diagnosis of Cancer, LLOYD F. CRAVER, M.D., Attending Physician, Memorial Hospital, New York City

Discussion: Karl F. Eschelman, M.D., Director, Tumor Clinic, Edward J. Meyer Memorial Hospital, Buffalo, New York

Advances in Surgical Treatment of Cancer, JOHN J. MORTON, JR., M.D., Professor of Surgery, University of Rochester School of Medicine, Rochester, New York

Discussion: George W. Cottis, M.D., President-elect, Medical Society of the State of New York, Jamestown, New York

Biopsy—Indications and Methods, J. HOWARD FERGUSON, M.D., Associate Professor of Pathology, Syracuse University College of Medicine, Syracuse, New York

Discussion: Rudolph J. Shafer, M.D., Director of Laboratories, Steuben County, Corning, New York

DINNER MEETING
6:15 P.M.

Principles of Radium and X-Ray Therapy, G. ALLEN ROBINSON, M.D., Director, Tumor Clinic, Flower and Fifth Avenue Hospitals, New York City; WALTER T. MURPHY, M.D., Radiologist, State Institute for the Study of Malignant Diseases, Buffalo, New York

X-Ray as a Diagnostic Aid in Cancer, BYRL KIRKLIN, M.D., Chief of Section on Roentgenology, Mayo Clinic, Rochester, Minnesota

ARKANSAS RADIOLOGICAL SOCIETY

On Oct. 1, 1941, the radiologists of Arkansas, meeting in Little Rock, organized the Arkansas Radiological Society. Meetings will be held every three months and also in connection with the annual meeting of the state medical society. The officers are: President, Fred Hames, M.D., of Pine Bluff; Secretary-Treasurer, J. S. Wilson, M. D., of Monticello.

USE OF X-RAYS IN THE MANIPULATION OF FRACTURES OF THE EXTREMITIES

The following Memorandum on the dangers encountered in the use of x-rays in the manipulation of fractures of the extremities,[1] issued by the British X-Ray and Radium Protection Committee, has been sent to the Editor by our British colleagues, Dr. G. Harrison Orton and Dr. Sydney Russ.

X-rays may be used for this purpose in two ways:

1. By screening or taking radiographs (which can now be processed very rapidly) when the surgeon believes he has attained correct position. Radiography should be the usual procedure adopted, and should involve no exposure of the hands or person to a direct beam of x-rays.
2. By actually manipulating the fracture under fluorescent screen control. This inevitably produces conditions of grave risk, and cannot be rendered safe, for the surgeon's hands must at times come into the direct beam of x-rays.

Only in exceptional cases should the second alternative be adopted, and then only on the responsibility and under the direction of the senior surgeon. Such work should never be delegated to juniors.

General Conditions: The operating room should be satisfactorily darkened. Lights should be dimmed as far as possible before screening, and no lights should face the surgeon.

The previous films of the case should be seen and memorised, and not scrutinised in a bright viewing box immediately before the manipulation. It is essential that the surgeon should prepare his eyes for screening. This takes a certain amount of time, which varies with the individual, but a minimum period of five minutes in the dark should be allowed for this. It can also be attained by using a single red light in the room or by the wearing of dark (preferably red) goggles for a period of some minutes before screening is begun.

The x-ray apparatus should be operated by a radiographer who has been specially trained for this work. He should be given definite instructions by the radiologist as to the limits within which he must use the apparatus. These limits should be governed by hard and fast rules, drawn up by the radiologist in conjunction with the surgeon in charge, and not in any circumstances to be varied if a surgeon finds himself in difficulty.

[1] The same considerations apply to the removal of foreign bodies under x-rays.

The radiographer should keep a log book recording the factors with actual times of screening involved. This log book should be signed by the surgeon after each operation, and the book should be inspected at regular intervals by the radiologist.

Operating Conditions: In general the recommendations of the British X-ray and Radium Protection Committee should be followed.

The tube-screen distance should not be less than 20 or more than 30 inches.

The apparatus should be set to give not more than 65 kv. For thin parts and children a lower kilovoltage will be adequate. *Three milliamperes is the maximum screening current that should be used.*

As a safeguard against excessive screening current, an automatic "cut-out" can be incorporated in most x-ray sets at a small cost, which will "cut out" if the selected factors are exceeded.

All apparatus should be shock-proof. An aluminium filter of 1 or 1.5 mm. should be fixed in the aperture of the x-ray tube so as to furnish a total filtration of not less than 2 mm. of aluminium. Cones or cylinders to limit the x-ray field are essential; these should be of such size that the illuminated area falls well within the limits of the fluorescent screen. An illuminated area of 5 inches circle, or 4 inches by 6 inches rectangle, is ample for this type of work. The radiographer should constantly watch to see that the x-ray beam is kept within the limits of the fluorescent screen. It is his duty to point it out to the surgeon if this essential factor is not complied with. If the x-ray tube and screen can be coupled to ensure this, it is a major safeguard which should be adopted.

An integrating time switch is useful. All exposures should be made with a timing device.

The fluorescent screen, faced with lead glass, should be of modern manufacture and in good condition. It must have handles that afford protection to the hands. A mechanical device for holding the screen is, however, preferable.

Under the above conditions screening and radiography between manipulations can be carried out with safety, provided always that the hands of the operator or of the assistant who holds the screen or films do not come within the direct beam.

Manipulation of fractures under screen observation should only be employed in very exceptional cases, preferably, if possible, with a radiologist in attendance. The time occupied in such manipulation should be strictly limited to one minute, and this should be accurately computed by the radiographer in attendance. It should be recognized that, even with one rapid manipulation with the hands in the direct beam, the safety tolerance dose may be considerably exceeded. *If the surgeon is willing to take this risk, it is entirely on his own responsibility.*

(*Note:* In the direct beam the exposed hands are subject to the daily safety tolerance dose in the prescribed conditions in about 2 seconds at 20 inches. At shorter distances, *e.g.*, when the reflecting fluoroscope is employed, the hands are nearer the tube and the dose is increased.)

The cryptoscope (the fluorescent screen fitted to the head and supplied with a hood) is a very dangerous device, and should never be used.

Operating conditions vary greatly in different hospitals, and it is suggested that each should draw up its own set of regulations. A list of specific safety instructions should be prepared and posted in the departments concerned.

CORRECTION

Ting, T. P.: A Simple Calculator for Roentgenographic Mensuration

In the article entitled "A Simple Calculator for Roentgenographic Mensuration," by T. P. Ting, in RADIOLOGY for August 1941 (**37:** 208, 1941), the curves in Figures 1 and 2 are unfortunately incorrectly labelled. Corrections should be made as follows:

In Fig. 1, "image-size in cm." should read "*object-size in cm.*"; "x = object-size in cm." should read "a = *image-size in cm.*"; "$x = 0, x = 1, x = 2, x = 3, x = 4, x = 5, x = 6$" should read "$a = 0, a = 1, a = 2, a = 3, a = 4, a = 5, a = 6$."

In Fig. 2, "object-size in cm." should read "*image-size in cm.*"; "a = image-size in cm." should read "x = *object-size in cm.*"; "$a = 0, a = 1, a = 2, a = 3$" should read "$x = 0, x = 1, x = 2, x = 3$."

In Memoriam

ALLEN HANSON BLAKE
1881–1941

Dr. Allen Hanson Blake of West Somerville, Mass., died early in December 1941, after a brief illness. Dr. Blake was born in Cambridge, sixty years ago, was graduated from Harvard Medical School in 1904, and spent the two following years in the Panama Canal Zone. From 1910 until his death he was on the staff of the Somerville Hospital. He was a member of the Radiological Society of North America and of the New England Roentgen Ray Society.

WALTER COX BARKER
1878–1941

Notice has been received of the death of Dr. Walter Cox Barker of Philadelphia on Dec. 26, 1941. Dr. Barker was born in 1878 and was a graduate of Hahnemann Medical College of Philadelphia, which he subsequently served as Consulting Roentgenologist. He was Radiologist to the Broad St. Hospital and was a member of the Radiological Society of North America and of the American Roentgen Ray Society

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

NASAL SINUSES. AN ANATOMIC AND CLINICAL CONSIDERATION. By O. E. VAN ALYEA, M.D., Assistant Professor, Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, Chicago. A volume of 262 pages with 82 illustrations. Published by The Williams & Wilkins Company, Baltimore, 1942. Price $6.50.

Book Reviews

THE 1941 YEAR BOOK OF RADIOLOGY. Diagnosis edited by CHARLES A. WATERS, M.D., Associate in Roentgenology, Johns Hopkins University; Assistant Visiting Roentgenologist, Johns Hopkins Hospital, and WHITMER B. FIROR, M.D., Assistant in Roentgenology, Johns Hopkins University; Assistant in Roentgenology, Johns Hopkins Hospital. Therapeutics edited by IRA I. KAPLAN, B.Sc., M.D., Director, Radiation Therapy Department, Bellevue Hospital, New York City; Associate Radiologist, Lenox Hill Hospital, New York City; Clinical Professor of Surgery, New York University Medical College. 440 pages and 486 illustrations. Published by The Year Book Publishers, Inc., Chicago, Ill., 1941. Price $5.00.

The 1941 Year Book of Radiology is the tenth of this excellent series. Like its predecessors, it presents in digest form a summary of the radiological literature of the world. Twenty-eight per cent of the articles are taken from journals published outside of the United States. Many come from clinical publications while the purely radiological journals are, as would be expected, fully represented.

In the diagnostic section the material is conveniently classified under the various systems of the body. A new section on Military Roentgenology has been added this year.

The section on therapy is also arranged on an anatomical basis, making for easy reference. In addition there are sections on Radiation Physics, Radiation Biology, and Radiation in General Medicine. A general summary of the status of radiotherapy during the year is given by one of the editors in the Introduction.

Illustrations reproduced from the original papers add greatly to the value of the work, and ample indexes of authors and subjects enhance its value as a reference book. It will find frequent use by busy radiologists. The accumulated volumes will be increasingly valuable when a cumulative index is available.

CLINICAL ROENTGENOLOGY OF PREGNANCY. By WILLIAM SNOW, M.D., Director of Radiology, Bronx Hospital, Roentgenologist-in-Charge, Harlem Hospital, New York City. A volume of 178 pages with 119 figures. Published by Charles C. Thomas, Springfield, Illinois, 1942. Price $4.50.

In the first third of this book the author describes the method of roentgenologic pelvimetry which he uses in his practice, discussing the positioning of the patient, roentgenologic technic, and the computation of the pelvic and cephalic diameters. His method, which is fairly simple, is a modification of the position method popularized by Thoms. To correct for the distortion of the various pelvic and cephalic diameters as measured on the films, computations are made using either a chart, which is reproduced in the book, or a special slide rule manufactured to the author's specifications by one of the x-ray equipment companies. Predictions concerning bony dystocia are based on these corrected measurements, together with a consideration of the general shape and contour of the pelvis as advocated by Caldwell, Moloy and their associates. Brief discussions of the Caldwell-Moloy classification and of the Turner "pelvic index" are included.

The second portion of the book is given over to a discussion of the roentgen visualization of the soft tissues in pregnancy. Determination of the site of implantation of the placenta, diagnosis of placenta previa, premature separation of the placenta, hydramnios, extra-uterine pregnancy, and tumors complicating pregnancy are all considered. Drawings and reproductions of roentgenograms add greatly to the value of the exposition.

In the last third of the book roentgenograms from twenty-two cases are reproduced to scale, together with the author's measurements and comments. This section is intended to serve as a practice ground where the reader, by making his own measurements, may become proficient in the interpretation of pelvimetric roentgenograms.

Although this book attains its purpose as a working manual of pelvimetry, it does so only for the method described. Anyone working in this field should not be satisfied until he has become familiar with the original works of Caldwell and Moloy, Thoms, Ball, and others whose writings are included in the bibliography, which, with a satisfactory index, completes the volume.

RADIOLOGICAL SOCIETIES OF NORTH AMERICA

Editor's Note.—Will secretaries of societies please cooperate by sending information to Howard P. Doub, M.D., Editor, Henry Ford Hospital, Detroit, Mich.

UNITED STATES

Radiological Society of North America.—Secretary, D. S. Childs, M.D., 607 Medical Arts Building, Syracuse, N. Y.

American Roentgen Ray Society.—Secretary, C. B. Peirce, Royal Victoria Hospital, Montreal, Canada.

American College of Radiology.—Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual Meeting, 1942, Atlantic City, N. J.

Section on Radiology, American Medical Association.—Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual Meeting, 1942, Atlantic City, N. J.

ARKANSAS

Arkansas Radiological Society.—Secretary-Treasurer, J. S. Wilson, M.D., Monticello. Meets every three months and annually at meeting of State Medical Society.

CALIFORNIA

California Medical Association, Section on Radiology.—Secretary, Joseph D. Coate, M.D., 434 Thirtieth St., Oakland.

Los Angeles County Medical Association, Radiological Section.—Secretary, Donald R. Laing, M.D., 65 N. Madison Ave., Pasadena. Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Society.—Secretary-Treasurer, L. Henry Garland, M.D., 450 Sutter St., San Francisco. Society meets annually during annual meeting of the California Medical Association.

San Francisco Radiological Society.—Secretary, J. Maurice Robinson, M.D., University of California Hospital. Meets monthly on third Thursday at 7:45 P.M., for the first six months at Toland Hall (University of California Medical School); second six months at Lane Hall (Stanford University School of Medicine).

COLORADO

Denver Radiological Club.—Secretary, Edward J. Meister, 366 Metropolitan Bldg. Meetings third Friday of each month at the Denver Athletic Club.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Secretary-Treasurer, Max Climan, M.D., 242 Trumbull St., Hartford. Meetings bimonthly, on second Thursday. Place of meeting selected by Secretary.

FLORIDA

Florida Radiological Society.—Secretary-Treasurer, Walter A. Weed, M.D., 204 Exchange Building, Orlando. Next meeting at annual meeting of State Medical Association, April 13–15, 1942, Palm Beach.

GEORGIA

Georgia Radiological Society.—Secretary-Treasurer, Robert C. Pendergrass, M.D., Prather Clinic Bldg., Americus. Meetings twice annually, in November and at the annual meeting of State Medical Association.

ILLINOIS

Chicago Roentgen Society.—Secretary, Chester J. Challenger, M.D., 3117 Logan Blvd. Meets at the Palmer House on the second Thursday of October, November, January, February, March, and April.

Illinois Radiological Society.—Secretary-Treasurer, William DeHollander, M.D., St. Johns' Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Secretary, Earl E. Barth, M.D., 303 E. Chicago Ave., Chicago.

INDIANA

The Indiana Roentgen Society.—Secretary-Treasurer, Harold C. Ochsner, Methodist Hospital, Indianapolis. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—Secretary-Treasurer, Joseph C. Bell, M.D., 402 Heyburn Bldg., Louisville. Meeting annually in Louisville, third Sunday afternoon in April.

LOUISIANA

Louisiana Radiological Society.—Secretary-Treasurer, Johnson R. Anderson, M.D., North Louisiana Sanitarium, Shreveport. Meets annually at same time as State Medical Society. Next meeting, New Orleans, April 1942.

Shreveport Radiological Club.—Secretary-Treasurer, W. R. Harwell, M.D. Meetings monthly on the second Wednesday, at the offices of the various members.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Secretary, Walter L. Kilby, M.D., 101 W. Read St. Meetings are held the third Tuesday of each month.

MICHIGAN

Detroit X-ray and Radium Society.—Secretary-Treasurer, E. R. Witwer, M.D., Harper Hospital, Detroit. Meetings first Thursday of each month from October to May, inclusive, at Wayne County Medical Society club rooms, 4421 Woodward Ave., Detroit.

Michigan Association of Roentgenologists.—Secretary-Treasurer, E. M. Shebesta, M.D., 1429 David Whitney Bldg., Detroit. Meetings quarterly by announcement.

MINNESOTA

Minnesota Radiological Society.—Secretary, John P. Medelman, M.D., 572 Lowry Medical Arts Bldg., St. Paul. Meetings quarterly.

MISSOURI

The Kansas City Radiological Society.—Secretary, P. E. Hiebert, M.D., 907 North Seventh St. (Huron Bldg.), Kansas City, Kansas. Meetings last Thursday of each month.

The St. Louis Society of Radiologists.—Secretary, Wilbur K. Mueller, M.D., University Club Bldg. Meets on fourth Wednesday of October, January, March, and May, at a place designated by the president.

NEBRASKA

Nebraska Radiological Society.—Secretary, D. A. Dowell, M.D., 816 Medical Arts Bldg., Omaha. Meetings third Wednesday of each month at 6 P.M. in either Omaha or Lincoln.

NEW ENGLAND

New England Roentgen Ray Society (Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island).—*Secretary,* Hugh F. Hare, M.D., Lahey Clinic, Boston, Mass. Meets monthly on third Friday at Boston Medical Library.

NEW JERSEY

Radiological Society of New Jersey.—Secretary, H. J. Perlberg, M.D., Trust Co. of New Jersey Bldg., Jersey City. Meetings at Atlantic City at time of State Medical Society and midwinter in Newark as called by president.

NEW YORK

Associated Radiologists of New York, Inc.—Secretary, William J. Francis, M.D., 210 Fifth Ave., New York City. Regular meetings the first Monday evening of the month in March, May, October, and December.

Brooklyn Roentgen Ray Society.—Secretary-Treasurer, Leo Harrington, M.D., 880 Ocean Ave. Meetings held the fourth Tuesday of every month, October to April.

Buffalo Radiological Society.—Secretary-Treasurer, Joseph S. Gianfranceschi, M.D., 610 Niagara St. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Ray Society.—Secretary-Treasurer, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse. Meetings are held in January, May, and October, as called by Executive Committee.

Long Island Radiological Society.—Secretary, Marcus Wiener, M.D., 1430 48th St., Brooklyn. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—Secretary, Paul C. Swenson, M.D., Presbyterian Hospital, New York, N. Y.

Rochester Roentgen-ray Society.—Secretary, S. C. Davidson, M.D., 277 Alexander St. Meetings at convenience of committee.

NORTH CAROLINA

Radiological Society of North Carolina.—Secretary-Treasurer, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meeting with State meeting in May, and meeting in October.

NORTH DAKOTA

North Dakota Radiological Society.—Secretary, L. A. Nash, M.D., St. John's Hospital, Fargo. Meetings by announcement.

OHIO

Ohio Radiological Society.—Secretary, J. E. McCarthy, M.D., Cincinnati. The next meeting will be held at the time and place of the annual meeting of the Ohio State Medical Association.

Cleveland Radiological Society.—Secretary-Treasurer, J. O. Newton, M.D., 13921 Terrace Road, East Cleveland. Meetings at 6:30 P.M. at the Mid-day Club, in the Union Commerce Bldg., on fourth Monday of each month from October to April, inclusive.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*Secretary-Treasurer,* Justin E. McCarthy, M.D., 707 Race St. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—Secretary-Treasurer, L. E. Wurster, M.D., 416 Pine St., Williamsport. The Society meets annually; time and place of next meeting will be announced later.

The Philadelphia Roentgen Ray Society.—Secretary, Barton R. Young, M.D., Temple University Hospital, Philadelphia. Meetings held first Thursday of each month at 8:15 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22nd St., Philadelphia.

The Pittsburgh Roentgen Society.—Secretary-Treasurer, Harold W. Jacox, M.D., 4800 Friendship Ave., Pittsburgh, Pa. Meetings are held on the second Wednesday of each month at 4:30 P.M., from October to June, at the Pittsburgh Academy of Medicine, 322 N. Craig St.

ROCKY MOUNTAIN STATES

Rocky Mountain Radiological Society (North Dakota, South Dakota, Nebraska, Kansas, Texas, Wyoming, Montana, Colorado, Idaho, Utah, New Mexico).—*Secretary,* A. M. Popma, M.D., 220 North First St., Boise, Idaho.

SOUTH CAROLINA

South Carolina X-ray Society.—Secretary-Treasurer, Malcolm Mosteller, M.D., Columbia Hospital, Columbia. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—Secretary-Treasurer, Franklin B. Bogart, M.D., 311 Medical Arts Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—Secretary-Treasurer, L. W. Baird, M.D., Scott and White Hospital, Temple.

VIRGINIA

Virginia Radiological Society.—Secretary, Charles H. Peterson, M.D., 603 Medical Arts Bldg., Roanoke.

WASHINGTON

Washington State Radiological Society.—Secretary-Treasurer, Kenneth J. Holtz, M.D., American Bank Bldg., Seattle. Meetings fourth Monday of each month at College Club, Seattle.

WISCONSIN

Milwaukee, Roentgen Ray Society.—Secretary-Treasurer, Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

CANADA

Section on Radiology, Canadian Medical Association.—Secretary, W. J. Cryderman, M.D., Medical Arts Bldg., Toronto.

Section on Radiology, Ontario Medical Association.—Secretary, W. J. Cryderman, M.D., 474 Glenlake Avenue, Toronto.

Canadian Association of Radiologists.—Honorary Secretary-Treasurer, A. D. Irvine, M.D., 540 Tegler Bldg., Edmonton, Alberta.

La Société Canadienne-Française d'Eléctrologie et de Radiologie Médicales.—General Secretary, Origène Dufresne, M.D., Institut du Radium, Montreal. Meetings are held the third Saturday of each month, generally at the Radium Institute, 4120 East Ontario Street, Montreal; sometimes, at homes of members.

CUBA

Sociedad de Radiología y Fisioterapia de Cuba.—Offices in Hospital Mercedes, Havana. Meetings are held monthly.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

The Head and Neck

The Chest

The Digestive Tract

The Skeletal System

Gynecology and Obstetrics

RADIOTHERAPY

Malignant Tumors

ROENTGEN DIAGNOSIS

Correlation of X-Ray and Autopsy Observations. Sydney J. Hawley. Pennsylvania M. J. **44:** 1534–1537, September 1941.

A study was made of 261 patients that came to autopsy, who had previously had various roentgen examinations. From the roentgenologist's point of view the correlation was gratifyingly high. No errors were detected in the following conditions: normal lungs, silicosis, passive congestion, atelectasis, carcinoma of the bronchus, mediastinal metastasis, pneumothorax, diaphragmatic elevation, drowned lung, mediastinitis, substernal thyroid, and sarcoid. Most of these conditions are so obvious that no errors should be made.

A number of the so-called errors of diagnosis were in cases where a definite diagnosis could not possibly have been reached. The majority were those of interpretation of the significance of the shadows. Many such errors can be eliminated by closer consideration of the clinical findings in any given case, and more frequent use of repeat and serial examinations. These additional roentgen-ray studies would also increase the specificity of roentgen interpretation.

The author concludes: "It appears that the x-ray examination leaves only little to be desired in the way of antemortem representation of gross morbid as well as normal anatomy." JOSEPH T. DANZER, M.D.

THE HEAD AND NECK

Radiation and Surgical Treatment of Malignancy in Paranasal Sinuses. W. L. Atkins. New Orleans M. & S. J. **94:** 19–23, July 1941.

The paranasal sinuses are the seat of a wide variety of tumors. The maxillary sinus is the most frequent site, followed by the ethmoid. The sphenoid and frontal sinuses are involved by extension, but frontal involvement is rare.

The choice of treatment of malignant neoplasms of the sinuses is based on the histologic nature of the growth. Tumors of highly cellular, anaplastic, embryonic type, that tend to metastasize quickly, as a rule respond poorly to surgery. It is in these types, therefore, that reliance is placed upon x-ray and radium therapy. Individualization of treatment is important and a decision must be made in each case as to whether cure or merely palliation is to be attempted. Close co-operation between the radiologist and surgeon is of paramount importance.

Some anaplastic tumors, lympho-epithelioma and Schneiderian carcinomas, are very radiosensitive and are successfully treated by x-radiation alone. They metastasize early, and the lymph drainage areas should be treated as intensively as the primary lesion, which is often very difficult to locate.

The slower growing carcinomas of the maxillary sinus are usually best treated by a preoperative course of x-ray therapy. Small multiple portals of entry are employed to utilize the crossfire radiation technic; the dosage is fractionated and treatment continued to skin tolerance. A total dosage of 2,000 to 2,500 r in air can safely be employed. This usually causes shrinkage of the tumor and after two or three weeks it can be removed with its bony extensions. Radium is applied at the time of the operation, packed in place with gauze, or later embedded in Stent or a Columbia paste mold. Filtration should be 1 mm. of platinum and dosage should be 3,000 to 4,000 milligram hours. The author has used platinum capsules but others have reported good results with radon seeds. The importance of radical removal of the tumor and of all invaded bone is stressed. Radiation cannot be expected to kill epithelial cells that have invaded the bone.

The author does not report his own results but concludes that the outlook in malignant tumors of the sinuses is grave, even in early cases. The late cases are almost always hopeless.

THE CHEST

The Inspissated Cavity. Arnold Shamaskin. Am. Rev. Tuberc. **44:** 310–322, September 1941.

The significance of the inspissated cavity is discussed and cases are presented to illustrate the evolution, behavior, and ultimate fate of this type of lesion. The only conclusive proof of a blocked inspissated cavity is a series of roentgenograms in which an area of decreased density in an earlier film shows in a subsequent film a fluid level, still later to be replaced by a homogeneous density occupying the same anatomical area of the lung field. If the changes occur in the reverse order, the proof is less conclusive, since the shadow may have represented a caseous bronchopneumonic focus which has become excavated.

An inspissated cavity appears on the x-ray film as a racket-shaped or circular shadow of more or less homogeneous density which is quite sharply demarcated from the surrounding lung tissue. The lung in the immediate vicinity of the shadow as a rule, but not invariably, is relatively clear or at least free from extensive progressive disease. The density of the shadow is similar to that of an area of caseous bronchopneumonia of the same dimension. Linear markings may be traced running from the pole of the inspissated cavity nearest the mid-line to the hilum of the lung. These apparently represent the draining bronchi. In most cases evidences of tuberculosis, past or present, in other parts of the lung are found. Coincidentally with the conversion of the area of decreased density into an area of increased density and the absorption of exudate around it, the clinical picture is that of a state of retrogression or arrest except in the presence of a progressive lesion elsewhere.

Inspissated cavities, especially when isolated, may resemble neoplastic metastases, but the latter are frequently multiple, and there is usually evidence of the primary lesion elsewhere in the body. No connection with the bronchial tree can be seen, and the lung is not, as a rule, the seat of an older tuberculous lesion. A benign neoplasm, a cyst, or a chronic pulmonary abscess may also be indistinguishable from an inspissated cavity.

The treatment of the inspissated cavity will depend upon its behavior and upon the condition of the rest of the lung. If it shows a tendency to open and close alternately, it should be treated, like any other cavity, by collapse therapy. If it shows no tendency to reopen, if the rest of the lung is either clear or shows healed lesions, and the patient presents no clinical or other evidences of activity, a watchful policy is best. It is important to make a distinction between cavity blockage with inspissation of content and cavity blockage with sputum retention, since the latter cases are characterized by sudden reduction or complete suppression of sputum accompanied by severe cough, chest pain, marked elevation of temperature, and other symptoms of toxemia. LESTER W. PAUL, M.D.

Pneumoperitoneum Supplementing Phrenic Paralysis. Arnold B. Rilance and Frederick C. Warring, Jr. Am. Rev. Tuberc. **44:** 323–334, September 1941.

An analysis is given of results obtained where pneumoperitoneum has been used to supplement phrenic paralysis in the treatment of pulmonary tuberculosis Pneumoperitoneum is slightly more difficult tech-

nically to administer than pneumothorax but has been found to be a safe procedure; it causes no more discomfort or inconvenience than pneumothorax and can be maintained in ambulatory patients. Of a series of 55 patients in whom pneumoperitoneum was induced to supplement phrenicectomy, slightly more than 50 per cent showed roentgenologic evidence of closure of cavity and in slightly less than 50 per cent either the sputum became negative or residual positive sputum could be explained by demonstrable bronchial ulcers.

In 18 of the cases (33 per cent) pneumoperitoneum was abandoned because of insufficient additional rise of the diaphragm. Elevation of the hemidiaphragm in mechanically effective cases averaged 2.8 cm. after phrenicectomy. Additional elevation following pneumoperitoneum averaged 4.1 cm. The authors believe that the combined procedures are of definite value when indicated. In cases unsuitable for phrenic paralysis in the first place, there is rarely much benefit derived from supplementary pneumoperitoneum.

LESTER W. PAUL, M.D.

"Armored Heart" with Secondary Stasis. L. Bischoff. Schweiz. med. Wchnschr. **71:** 1074, Sept. 13, 1941.

Brief report of a case of extensive pericardial calcification in a man of 62, which by preventing the expansion and dilatation of the heart led to stasis in the vena cava with symptoms of a pseudocirrhosis and ascites. Other members of the family had died of conditions with edema. The author was considering the possibility of operation in spite of the patient's age as the general and cardiac conditions were otherwise satisfactory. Roentgenograms illustrating the calcification are reproduced. LEWIS G. JACOBS

THE DIGESTIVE TRACT

Small Intestine in Vitamin B Deficiency. Ross Golden. J. A. M. A. **117:** 913–917, Sept. 13, 1941.

The author reviews the observations which have been made on the small intestine in a series of more than 100 cases in which some deficiency condition appeared to play either a major or a minor part. Deficiency states may conveniently be divided into two groups, primary and secondary. A primary deficiency is one which occurs without obvious cause, in which a chronically deficient diet may be a factor, *e.g.*, sprue or pellagra. A secondary deficiency is one which is associated with, or perhaps caused by, recognizable disease of the alimentary tract, such as cancer or regional enteritis.

To demonstrate abnormalities in the small intestine the author makes what he terms a "small intestine study." Roentgenograms of the abdomen are taken at approximately half-hourly intervals after the ingestion of a barium sulfate suspension in the morning during fasting; 4 ounces of chemically pure barium sulfate in physiologic solution of sodium chloride made with distilled water are used. Films are developed and inspected immediately, and the routine is modified if it seems advisable. A series is concluded within five or six hours, occasionally sooner.

The abnormalities of the small intestine associated with vitamin deficiency appear to vary with the severity and duration of the disorder. Improvement in the roentgen appearance under treatment usually lags behind the clinical improvement.

The following observations are made.

Hypermotility: In the earlier stages of the deficiency the barium sulfate may reach the lower part of the small intestine in a quarter of an hour and enter the cecum in less than half an hour. Sometimes the jejunum alone shows marked hypermotility.

Hypertonicity: Reduction of the lumen to one-half, one-fourth, or even less, is often noted in the earlier stages. Relaxation ahead of the wave of contraction is sometimes absent.

Hypomotility: This is noted in the more advanced stages.

Hypotonicity (Dilatation): Dilated loops are characteristically seen in the well advanced stages, usually called non-tropical sprue and adult celiac disease.

Abnormal Segmentation: Areas of spasm of variable length narrow the lumen in places, sometimes completely expelling the opaque material from the contracted area and giving the appearance of discrete masses. Movement back and forth is often seen, with little forward progression of the barium. Segmentation is usually more pronounced in the middle third of the intestine and in the less advanced stages of the condition.

Scattering Effect: In some cases, as the main masses of barium sulfate pass along, small irregular masses linger behind. These shadows remain for some time scattered along the course of the intestine, usually in the upper loops. This effect seems to be due in part to a disturbance in the function of the circular muscle and in part to a disturbance in the movement of the mucosa which depends on the muscularis mucosae.

Gas and Fluid Levels: Gas may be present in considerable amounts. Gas and fluid levels may even suggest ileus (Kantor).

Mucous Membrane: The mucosal folds may be greatly reduced in number or may be completely obliterated. Sometimes they are "exaggerated."

The abnormal motility phenomena—segmentation and changes in tone and motility—are thought to be due to defective response of the longitudinal and circular coats of the tunica muscularis to the normal stimulus of intestinal contents. Abnormalities in the mucosal contours must be attributed to inability of the muscularis mucosae to respond as usual to normal stimuli.

Except in cases of the most advanced, severe vitamin deficiency appropriate treatment is followed by improvement in the function of the small intestine, as manifested by roentgen examination. In some cases the patient may recover completely as far as symptoms are concerned, but persistent abnormalities may remain in the middle loops.

Slow emptying of the stomach is frequently found in well marked primary deficiency states. The author has observed it also in some early cases and has found that it disappears promptly following vitamin therapy.

In fatal cases, atrophy of the tunica muscularis and of the mucosa, edema, round-cell infiltration and fibrosis of the submucosa and ulceration have been described. Degeneration of nerve cells in both the myenteric and the submucosal plexuses has also been found.

The changes described in the roentgen appearance of the small intestine in cases of vitamin deficiency occur in other conditions as well, the most important being diseases associated with hypoproteinemia (*e.g.*, nephrosis) and with disorders of the liver. The intestine of the normal newborn infant also shows a typical "deficiency pattern." The author suggests a possible common mechanism for these phenomena and names the intramural nervous system as a possible, if not the most probable, medium through which these effects are produced.

It must be borne in mind (1) that a deficiency state often exists with other organic disease, (2) that the demonstration of a "deficiency pattern" does not rule out other organic disease, (3) that the symptoms of a deficiency state may be obscured by those of the primary organic disease, *e.g.*, peptic ulcer, and (4) that the symptoms of a deficiency state may be the only manifestation of the underlying organic disease, *e.g.*, diffuse lymphosarcoma of the intestine. From the

clinical standpoint, it seems to be agreed that the whole vitamin B complex is necessary to relieve the abdominal symptoms.

The differentiation of the various fundamental causes of abnormalities of the small intestine is impossible at present. For this reason the demonstration of the described changes on roentgen examination can be taken only as suggesting certain possibilities. In the author's cases the most common cause appears to be a vitamin B deficiency. Evidence of this is the clinical relief with the parenteral or the oral administration of liver extract or the vitamin B complex. It is considered possible, however, that under certain circumstances the less marked forms of the "deficiency pattern" might be due to allergy.

The described changes can be taken as strong evidence of a nutritional disturbance, usually one in which vitamin deficiency plays a part but which might be due to hepatic disease or primarily to hypoproteinemia. One case is cited in which changes progressed to severity in spite of oral administration of the vitamin B complex, only to regress rapidly toward the normal after the institution of parenteral vitamin therapy. Investigation of another case showing moderate changes in the small intestine led to the discovery of the presence of multiple myeloma. In spite of the non-specificity of these intestinal disturbances, they are proving helpful in the search for the cause and relief of obscure abdominal symptoms. It is worthy of emphasis that knowledge of the physiology of the small intestine is at present incomplete.

[In connection with this paper the reader will find the Carman Lecture, delivered by the author before the Radiological Society of North America, of interest (Radiology **36:** 262, 1941), especially as it includes a review of the normal anatomy and physiology of the small intestine.—ED.] CLARENCE E. WEAVER, M.D.

Tumefactive Lesions of the Small Intestine. C. Allen Good. J. A. M. A. **117:** 923–926, Sept. 13, 1941.

The granulomas or non-neoplastic tumefactions of the small intestine are due in most instances to chronic non-specific enteritis or to tuberculosis. Those caused by other inflammatory conditions are extremely rare The most frequently encountered benign blastomas are leiomyoma and adenoma. Most important of the malignant neoplasms is adenocarcinoma; leiomyosarcoma, fibrosarcoma, and lymphoblastoma also occur.

Two methods for the examination of the small intestine are available. Examination of the terminal portion of the ileum, the site of the majority of the non-neoplastic tumefactions, may be accomplished at the time of the administration of a barium sulfate enema. Reflux of the opaque solution into the ileum permits a satisfactory examination of this portion of the bowel. The remainder of the small intestine is examined best by means of the opaque meal. Frequent roentgenoscopic observations should be made in addition to the customary roentgenograms.

Tumefactive lesions of the small intestine usually manifest themselves roentgenologically in one or more of three ways: by producing a "filling defect," by obstruction, by intussusception.

The deformity, or "filling defect," produced by a tumefactive lesion may be marginal, encircling, or central. An analysis of the morphologic features of this deformity often leads to an accurate estimation of the histologic type.

The gross pathologic features of tuberculosis and non-specific enteritis of the small intestine are strikingly similar. Roentgenologically they are characterized by involvement of fairly long segments of bowel. The deformity produced is that of constriction of the lumen. Demarcation between involved and uninvolved intestine is gradual. The mucous membrane may or may not be destroyed by ulceration and the mucosal pattern correspondingly changed. The presence of an extra-alimentary focus of tuberculosis is an aid in the differentiation between non-specific enteritis and tuberculosis of the bowel.

In contradistinction to the long deformity produced by an inflammatory process, that caused by a neoplasm is relatively short and sharply demarcated in the long axis of the bowel. This is true whether the neoplasm is benign or malignant. It must be emphasized, however, that there are no reliable roentgen signs of benignancy once the diagnosis of blastoma is established.

The various benign neoplasms of the small intestine exist chiefly as pedunculated intraluminal growths or as intramural submucous nodules. All may cause intussusception, and when such is the case they exhibit no characteristic features by which they may be distinguished.

Pedunculated intraluminal tumors produce central filling defects in the column of the opaque meal. As they grow larger they tend to cause partial obstruction. Adenoma, leiomyoma, lipoma, and fibroma are the most important varieties.

Intramural, submucosal tumors exhibit marginal defects with smooth contours. The mucosal pattern is often preserved. The most common tumor in this category is leiomyoma, and this type of growth may be suspected when the marginal defect contains a central niche characteristic of ulceration. These tumors are prone to break down, ulcerate, and bleed while remaining small in size.

Chief among the diagnostic signs of malignancy is the obliteration of the mucosal pattern throughout the extent of the lesion. The deformity is short and sharply limited; it may be marginal or encircling. The demarcation of the involved area is abrupt. The involved segment is stiffened. The luminal aspect of the tumor is marked by rough, jagged contours. There may be partial or complete obstruction, and in rare instances intussusception. The most common malignant tumor which involves the small intestine is adenocarcinoma. This usually produces an annular or polypoid defect in the barium column. Partial obstruction with dilatation of the bowel aborally is common.

The various forms of sarcoma, because they arise from tissues beneath the mucous membrane, cannot be distinguished from their benign counterparts until such a time as the lesions, because of their size, rapid growth, or tendency to infiltrate, exhibit the roentgenologic characteristics of malignancy.

It is emphasized that the frequency with which lesions of the small intestine are located and identified depends solely on the care taken by the examiner while viewing and manipulating all segments of the bowel during roentgenoscopy.

CLARENCE E. WEAVER, M.D.

Omental Adhesions Syndrome. Postoperative Dysfunction of the Transverse Colon. James C. McCann. Surg., Gynec. & Obst. **72:** 707–721, April 1941.

The author believes that omental adhesions produce a distinct clinical syndrome quite separable from the ill-defined category of postoperative adhesions in general. This syndrome is based on dysfunction of the transverse colon induced by the pathologically fibrosed adherent omentum fixed under tension to a low abdominal incision. The symptoms are of three types: (1) reflex, consisting of epigastric distress after eating, nausea, regurgitation, and eventually vomiting; (2) symptoms referable to the colon, which appear to dominate the picture, namely, attacks of sharp colicky pain, intractable constipation, distention, pain accompanying bowel movements whether spontaneous or

induced by an enema, and relief after dejection; (3) peritoneal symptoms, chiefly discomfort or colicky pain due to constant tension and intermittent traction on the colon as a result of physical exertion. The general clinical picture may be one of explosive onset of symptoms, suggesting an acute abdominal emergency but gradually abating, one of chronic recurrent colic, or one of repeated attacks of apparent subacute intestinal obstruction with no roentgenologically demonstrable cause.

Postoperative omental fixation is not necessarily productive of symptoms. Where, however, the adherent omentum undergoes progressive fibrosis and shortening, displacing the colon, dysfunction ensues with the production of the syndrome described. In 20 of a series of 23 consecutive cases encountered by the author over a period of seven years complete preoperative and postoperative roentgen studies were made, yielding evidence of colonic dysfunction in 14 or 70 per cent. Integration of the clinical, roentgenologic, and physiological data explain the dysfunction as due to the irritating effect of the omental adhesions on the colon, enhanced by the stimulus of normal functional activities. Roentgenograms taken when traction was purposely produced on the colon *via* the omentum showed spasm which was not present in the absence of traction; 30 per cent of the preoperative roentgenograms showed dilatation and diminishing haustrations, called "colitis"; 20 per cent showed a toneless elongation of the transverse colon which disappeared following release of the adhesions, apparently from restored tone; 30 per cent showed heightened irritability as manifested by local or segmental spasm or general hyperperistalsis; 25 per cent showed evidence of delayed emptying of the colon; 15 per cent showed late retrograde transport of barium.

The syndrome occurs predominantly in women. In the author's series infection did not seem to play an impressive rôle, though Muller and Rademaker (Arch. Surg. **26:** 208, 1933) regarded this as an important factor in postoperative adhesions.

Measures for prevention of omental adhesions are of the first importance. Treatment consists in their surgical release, with appropriate methods to prevent recurrence. Surgical failures, however, are not unknown.

An illustrative case is reported and numerous roentgenograms are reproduced.

Polyps of the Rectum and Colon in Infants and in Children. Roger L. J. Kennedy. Am. J. Dis. Child. **62:** 481–488, September 1941.

During the years 1925 to 1940, inclusive, at the Mayo Clinic, 49 infants and children with polyps of the rectum and colon were observed. Nearly half of the patients were four to six years old. Thirty were boys and nineteen were girls. Blood in the stool was the most frequent symptom, occurring in 43 of the 49 patients. Other symptoms in the order of occurrence were protruding mass, abdominal pain, diarrhea, and mucus in stool. A small group was symptomless or showed other evidence of the disease. The majority of patients were seen after symptoms had been present for six months to three years.

Roentgen examination in addition to proctoscopy was carried out in 32 of the 49 cases. Three patients had diffuse polyposis and 2 patients had pseudopolyps accompanying colitis. The remaining 27 patients had one to seven polyps distributed as follows: rectum only, 15; colon only, 4; both rectum and colon, 8.

Treatment was principally by fulguration, although ligation, excision, and resection were employed in some cases. One patient also received x-ray therapy. No treatment was given in 9 cases, including 2 of diffuse polyposis.

Tissue for pathological examination was obtained in 18 cases, 10 of which showed varying degrees of malignancy.

The authors conclude that all patients with rectal polyps should be carefully examined roentgenologically for polyps in the colon, and that all adenomatous polyps, except those found in certain cases of ulcerative colitis, have carcinomatous potentialities and should therefore be removed or destroyed.

GEORGE M. WYATT, M.D.

Foreign Bodies in the Biliary Tract: Report of Case with Tabular Review of Literature. Fred H. Bowen. Arch. Surg. **43:** 458–461, September 1941.

A report of a case in which 2 inches of a No. 20F catheter broke off in the common duct after a cholecystectomy. Four years later a recurrence of symptoms led to reoperation; the catheter, covered with and occluded by calcareous deposits, was removed and the patient recovered. Eight other cases found in the literature are tabulated for comparison.

LEWIS G. JACOBS, M.D.

Postoperative Cholangiography. N. F. Hicken, Q. B. Coray, and J. F. Orem. Rocky Mountain M. J. **38:** 709–713, September 1941.

In the opinion of the authors, the postoperative cholangiogram determines the patency of the bile ducts, measures the degree and rapidity of ductal repair, and designates the proper time for removal of the drainage tubes.

Such intraductal lesions as "elusive gallstones," plugs of inspissated mucus, blood clots, detached fragments of liver, strictures, neoplasms, and compressive occlusion from an inflamed and swollen pancreas, have been brought to light by means of cholangiograms. In cases in which postoperative jaundice had developed and in which it was feared that the common duct might have been traumatized or ligated the cholangiogram demonstrated the duct to be patulous.

It is generally assumed that the spontaneous closure of an external biliary fistula is ample proof that the common bile duct harbors no obstructive lesion. When the sinus tract formed by the removal of the drainage tube heals, the surgeon feels that his work has been successfully accomplished. Recently the authors have obtained cholangiographic evidence which contradicts these concepts. In three different patients the external biliary fistula healed spontaneously in spite of the fact that the cholangiograms outlined calculi which were incarcerated within the ampulla of Vater. The obstruction was incomplete.

The authors' experience suggests that every patient submitting to a cholecystostomy or choledochotomy should have a thorough cholangiographic study before the drainage tubes are removed.

The solution found preferable by the authors has been diodrast 70 per cent. Removal of gas from the gastro-intestinal tract by gentle catharsis or enemas facilitates examination. With the patient supine, the area from the fourth thoracic vertebra to the fourth lumbar is used for centering. In asthenic patients the tube is angled to the right so as to avoid superimposing the common duct shadow on the spine. Films may be made at the bedside or on the operating table with a portable unit.

The diodrast is heated to body temperature. The amount injected varies from 15 c.c. to 125 c.c. Fluoroscopy is often desirable, though not necessary.

If a first film shows the solution to have escaped into the duodenum without outlining the hepatic ducts the procedure is repeated. This time counter pressure is made over the ampulla of Vater, thus occluding the outlet of the common duct and forcing the fluid proximally. In general, fluid is injected until

some discomfort suggesting increased intraductal pressure is complained of by the patient.

If the films reveal an obstruction of the common duct, serial films are taken at ten-minute intervals. At times there will seem to be an ampullary obstruction but subsequent films reveal the sphincter of Oddi to be open, for the contrast medium is seen in the jejunum. This means that there was a temporary contraction of ductal sphincter, biliary dyssynergia, which blocked the choledochus, but as soon as it relaxed the diodrast passed into the duodenum. In all cases in which an ampullary obstruction is encountered, a tablet of nitroglycerine, 0.01 grain, is placed under the tongue and another film taken. Sphincterismus may thus be ruled out. PERCY J. DELANO, M.D.

THE SKELETAL SYSTEM

Varus Deformity of Ankle Following Injury to Distal Epiphyseal Cartilage of Tibia in Growing Children. Gerald G. Gill and Leroy C. Abbott. Surg., Gynec. & Obst. **72:** 659–666, March 1941.

The authors attribute varus deformity of the ankle to a specific injury of the medial malleolus, namely, the fracture-separation which may follow adduction injuries in growing children. Premature cessation of growth in the medial portion of the distal epiphyseal plate of the tibia as a result of such injury, while growth from the uninjured lateral portion continues, produces the varus deformity with shortening of the tibia. The mechanism whereby this occurs is explained and a case in a 15-year-old boy is recorded to illustrate the problems presented and their solution.

The injury in the case reported occurred five years earlier, and progressive deformity had been present for three years. Operation, carefully planned by means of x-ray films and dissections on the cadaver, consisted in first lengthening the tendons of the tibialis anticus, extensor hallucis, and extensor digitorum communis, and the Achilles tendon, followed by a very low, oblique osteotomy passing through the region of deformity distally and laterally and wedged open on the medial side, the wedge being filled with flakes of cancellous bone obtained from the ilium. A specially devised lengthening apparatus was then applied. Within a month full correction had been obtained but traction was continued for another month. A cast was then applied while the foot was still in the apparatus and a walking iron furnished support for about two months longer. Teleroentgen examination at the end of this period—four months after operation—showed a lengthening of the medial side of the tibia by three-fourths of an inch. Meantime there had been slight growth of the uninvolved tibia. In order to minimize the difference in length of the two extremities, which it was thought might increase with growth, a second operation was undertaken to close the upper epiphyses of the tibia and fibula of the normal left leg, but some doubt is expressed as to the effectiveness of this measure in the present case, as the skeletal maturation was already rather far advanced.

The patient was last seen fourteen months after operation, at which time correction had been maintained and he had full use of the foot and ankle, even to the extent of participating in such active sports as football and basketball.

While a good result was obtained in this case in spite of the long neglect, the authors urge repeated radiological check-ups of all fresh fractures through the medial portion of the distal epiphysis of the tibia, and the application of simple corrective procedures at the first evidence of deformity. An area of fusion between the medial malleolus and the diaphysis, with disappearance of the clear area occupied by the epiphyseal cartilage plate, is the earliest sign of trouble. At this time simple fusion of the lateral growing portion of the distal epiphysis of the tibia and the distal epiphysis of the fibula of the affected leg will result in clinical cure, as the other joints of the foot can easily compensate for the minor tilt already present. Attendant fusion of the distal epiphyses of the tibia and fibula of the normal leg will then assure practical equality in the length of the legs at full growth. In this manner, the patient will be saved not only the discomfort of the developing deformity but a major operative procedure in the future.

Arthrograms of Hip Joints of Children. Erik Severin. Surg., Gynec. & Obst. **72·** 601–604, March 1941.

To the comparatively meager literature on hip joint arthrography the author adds a study of the arthrograms of hips of normal children and of specimens from infants stillborn at full term. The specimens, prepared by injection of a mixture of cinnabar and celloidin, were examined roentgenographically and subsequently dissected for comparison with the roentgen films.

Arthrograms are reproduced showing the ribbon-like collection of contrast medium around the neck of the femur; the circular impression produced medial to this by the "orbicular zone"; and in the upper part of the joint the impression by the "fibrocartilaginous limbus," which the author likens to a ploughshare.

Normally the quantity of contrast medium between the femoral head and the acetabulum is very small, except medially. Here the contrast material is collected partly in the fossa acetabuli—*i.e.*, medial to the ligamentum transversum—partly in the relatively generous capsule pocket, which is to be found immediately outside the ligamentum transversum. The pointed fibrocartilaginous limbus merges into the ligamentum transversum at the incisura acetabuli. Sectionally the ligamentum transversum is rounded, and on account of its extension mainly in a dorsoventral direction seems, in pictures taken in a dorsoventral plane, to appear in the contrast medium as a rounded impression.

The quantity of contrast medium which lies in the fossa acetabuli and which forces its way down medial to the ligamentum transversum, is parted at its lower tip into two prongs—a ventromedial and a dorsolateral, the anatomical foundation of which are the two pouches which are designated by the author as recessus ventralis fossae acetabuli and recessus dorsalis fossae acetabuli.

In cases of congenital dislocation of the hip, one or more of the arthrographic details noted may be distorted or obliterated through more or less extreme secondary changes.

Fractures of Both Bones of the Forearm in Children. Boardman M. Bosworth. Surg., Gynec. & Obst. **72:** 667–669, March 1941.

In a period of seven years 50 children of sixteen years or less were treated in the outpatient department of St. Luke's Hospital (New York) for shaft fractures of both bones of the forearm. Thirty-two of these were traced, being followed for periods varying from a year and five months to over seven years. Twenty-five of the 32 were treated by closed reduction with excellent anatomical and functional results in 21, and good or fair results in the other 4. So satisfactory are the results of such conservative treatment that operation need seldom be resorted to. The present series of 50 cases includes 4 open reductions, which the author admits is too high an operative rate. All these patients were followed. Satisfactory results were obtained in 3 cases and a good clinical result in the fourth despite a non-union and pseudarthrosis of the ulna. Roentgenograms are reproduced to show some of the results obtained.

Juvenile Kyphosis. John T. Hodgen and Charles H. Frantz. Surg., Gynec. & Obst. **72:** 798–806, April 1941.

Juvenile kyphosis occurs as the result of abnormal functional demands upon a normal or abnormal back or, in a small group of cases, as the result of normal functional demands upon an abnormal back. It is of more frequent occurrence in girls, of the asthenic type, with onset usually at ten or eleven years of age. While there is much controversy as to the pathology, the authors summarize the situation with the statement that "destruction of the growth zone or epiphyseal cartilage zone may be caused by herniations of the intervertebral disc with resultant decrease in growth anteriorly in the vertebral body. While we have seen clinical kyphosis in the adolescent without radiological evidence of nuclear prolapse and again multiple nuclear herniations without kyphosis, we are sure that the nuclear herniations and cessation of growth of the body anteriorly are important considerations in the development of juvenile kyphosis."

Poor posture and pain in the back or fatigue are the chief complaints. The back is rounded, with limited mobility, and the head is carried forward, thus accentuating the arcuate curve of the dorsal spine. The roentgen criteria necessary for diagnosis are (1) an increased dorsal curve; (2) wedging of the vertebral bodies due to arrested growth anteriorly; (3) diminished intervertebral disc space; (4) saw-toothed serrations of the anterior borders of the vertebral bodies; (5) nuclear herniations into the vertebral bodies; (6) paravertebral thickening along the lateral borders of the spine in the affected area. In the late stages one may see secondary arthritic changes. The close approximation of the vertebral bodies resulting from the collapsed disc may allow bridging. Lipping of margins may develop.

Treatment consists in rest, physiotherapy, and temporary spinal supports, and is important if permanent deformity and chronic backache in later life are to be avoided.

Congenital Lumbosacral Defect. John G. Sinclair, Norman Duren, and J. C. Rude. Arch. Surg. **43:** 473–478, September 1941.

Two cases are recorded, one in a 9-year-old boy and one in a 60-mm. fetus, in which complete agenesis of the lumbar spine, sacrum, and coccyx was observed. In the living child neurological and muscular defects in the lower extremities were also present. The two ilia articulated, as did also the twelfth ribs. The authors believe this defect is due to faulty intervertebral planes at the time the primordia of the vertebral bodies are laid down. The extensor muscles dislocate the centra obliquely, the cartilages compressing the aorta and rupturing the muscles and nerves or destroying the ventral half of the cord. LEWIS G. JACOBS, M.D.

Radiological Aspect of the Bone Lesions in Mycetoma. Alejandro Castanedo K. Rev. de cir. (Mexico) **13:** 33–44, July 1941.

Madurella mycetomi, the fungus that in man causes mycetoma, better known as Madura foot, affects other parts of the skin as well as the feet. In an essay on the localization of these lesions in bones, the author states that the roentgen diagnosis is not of great importance as the complication appears late. He nevertheless believes that the subject has been neglected and is entitled to further observation. He quotes from other writers and records his own findings.

Cases are of three types. In the first group the predominating lesions are destructive, forming small areas of lesser density bounded by sclerosing osteitis, giving the bone a cystic appearance similar to the findings in osteitis fibrosa cystica. In this type bone destruction predominates and there is little, if any periosteal reaction. The second group shows a marked periosteal reaction extending far beyond the site of the primary lesion, subperiosteal changes, and ray-like projections of bony spicules, similar to those seen in osteogenic sarcoma. Although destructive lesions occur, the bone does not present the cystic appearance of group one. Cases of the third group do not show any specific changes. They represent the beginning of a process that will correspond at its termination to the types previously described. During this stage the periosteal reaction with the resulting ray-like projections of bone is initiated.

Osseous infection occurs by continuity. It is therefore logical to suspect that the initial lesion is periosteal, followed by response of the deeper bone tissues that form the ray-like projections. As the infection progresses, areas of bone destruction will occur that eventually are surrounded by sclerosed bone. The final roentgen image, therefore, presents a combination of the findings in the first and second groups.

A. MAYORAL, M.D.

Synovial Sarcomas in Joints, Bursae, and Tendon Sheaths. A Clinical and Pathological Study of Sixteen Cases. Dominic A. De Santo, Robert Tennant, and Paul D. Rosahn. Surg., Gynec. & Obst. **72:** 951–981, June 1941.

Sixteen cases of synovial sarcoma personally observed by the authors are reported. These tumors originate in synovial tissue and are thus found in joints, para-articular bursae, and tendon sheaths. They may be encapsulated, circumscribed, or diffuse, all three of these types being represented in the authors' series. The knee is the most usual site, being involved in 9 of the authors' 16 cases and 18 of a collected series of 37. The symptoms are neither striking nor characteristic. Pain, tumor, dysfunction, and swelling were observed, but none of these was invariably present.

It has been said that x-ray films of synovial sarcoma are usually negative, but to this the authors take exception. X-ray examination was made in 13 of their cases, and in 10 of these a soft part tumor near a joint was visualized. In 4 cases the tumor showed irregular calcification which facilitated x-ray visualization. In 2 cases the tumor was overlooked on first examination but was readily discovered on review of the original films. One case showed increased intra-articular soft part thickening five years before the diagnosis of synovial sarcoma was established, but this was not regarded with suspicion.

Closer attention to soft tissue detail should lead to the more frequent diagnosis of these tumors roentgenographically. Not unlikely, pneumarthrography will improve x-ray diagnosis. In two of this series extensive destruction of the bones comprising the knee was found without confirmatory x-ray evidence. For a more comprehensive review of the roentgenographic features of these tumors reference is made to a paper by Raymond W. Lewis (Am. J. Roentgenol. **44:** 170, 1940).

Amputation is the treatment of choice for synovial sarcomas, though in localized or encapsulated tumors radical excision followed by intensive irradiation may be justifiable. In cases thus treated the first evidence of recurrence calls for amputation. Eight of the authors' patients received intensive deep x-ray therapy after local excision, but none escaped recurrence or metastasis. One patient of this group, however, lived ten years before death from metastases.

The authors discuss at length the histologic features of these growths and reproduce numerous photomicrographs, as well as roentgenograms. Frequent reference is made to Berger's comprehensive study (Am. J. Cancer **34:** 501, 1938).

GYNECOLOGY AND OBSTETRICS

Accuracy of Roentgen Estimates of Pelvic and Fetal Diameters. A. Louis Dippel and E. Delfs. Surg., Gynec. & Obst. **72**: 915–922, May 1941.

This paper seeks to evaluate the precision of several of the better known methods of pelvimetry and cephalometry, with the idea that such an evaluation may lead to a wider utilization of these procedures.

The various methods are described and the authors conclude that each has an accuracy which is generally adequate for clinical purposes. In their hands the Hodges graphic, the precision, and the stereoroentgenometric methods have proved almost equally good in the production of precise estimates of maternal pelvic and fetal diameters. The footpoint method has shown variations in precision but has very definite possibilities. The Thoms and the Hodges lateral methods for measurement of anteroposterior pelvic diameters are generally quite accurate, but correct placement of the lead ruler in the former is highly essential to precision, and pelvic asymmetry precludes consistent precision with either method. The Thoms inlet view furnished precise estimates of transverse pelvic diameters only when the authors were fortunate in approximating the level of the pelvic inlet in the selection of the posterior external bony landmark. Two mathematical procedures for the production of precise estimates from stereoscopic films taken by a non-precision x-ray machine are given.

Roentgenological Survey of the Pelvis. Herbert Thoms and Hugh M. Wilson. Yale J. Biol. & Med., **13**: 831–839, July 1941.

The authors present an excellent description of their present technic in roentgen pelvimetry. The method is simple, and in daily practice should represent a minimum of time and inconvenience. Two views are taken: (1) a lateral projection of the pelvis with the patient standing in the upright position, and (2) a projection of the inlet of the pelvis. In this latter projection the inlet of the pelvis is horizontal to the film.

The authors believe that these two views are essential for complete evaluation of the pelvic capacity and information concerning the various planes and contours. They divide the pelvis into three portions: the pelvic inlet, the mid-plane, and the outlet.

Those interested in the technical details and method of interpretation should read the original article.

ANDREW H. DOWDY, M.D.

RADIOTHERAPY

MALIGNANT TUMORS

Treatment of Cancer of the Larynx. Chevalier L. Jackson. South. M. J. **34**: 243–254, March 1941.

In the decision between surgery and irradiation for the treatment of laryngeal cancer the location and extent of the lesion have usually been considered the most important factors, with the degree of mobility or fixation of the vocal cords also playing a rôle.

The author uses protracted fractional irradiation in cases unsuitable for laryngofissure in which laryngectomy is contraindicated by the patient's age, temperament, or physical condition; in cases in which the lesion has reached the posterior extremity of the cord, but the cord is not fixed; in "inoperable cases," in which the growth is extrinsic by origin or extension, or in which cervical metastases are present.

Whatever procedure is employed, the prognosis depends in some measure on the histopathology of the growth, and this the author believes should be taken into consideration in the selection of treatment in borderline cases. He says: "In the less differentiated, higher grade tumors we should feel inclined toward more radical surgery, or toward irradiation, rather than toward conservative operations, but at the same time these tumors are to be regarded as of poor prognosis by any method of treatment."

The technic of irradiation for an "average case" is as follows: 125 r daily to each side of the neck for twenty-five treatments, the skin portal measuring 7 to 10 cm., and the skin target distance 50 cm. The factors commonly used are 180 kv., 8 ma., 2 mm. Cu and 1 mm. Al. The dosage is 10.6 r per minute for 11.8 minutes on each side, the total dose to each side being 3,000 r or slightly more. In the presence of node metastases fractional irradiation is supplemented by the implantation of radon. Additional irradiation may be given after a year if indicated.

Statistically the results of surgical treatment are superior to the results of irradiation. In a series of 37 cases treated by irradiation alone, a three-year cure was obtained in 45 per cent, but this, the author warns, affords little assurance of a permanent cure, as recurrence may take place after five or more years. So far as the voice is concerned the results of irradiation are excellent, but this should not be given undue weigh when other indications are for surgical extirpation.

Carcinoma Erysipelatodes. Subepidermal Lymphatic Metastases Confused with Operative Sequelae. Mortimer R. Camiel and Herman Bolker. Surg., Gynec. & Obst. **72**: 635–641, March 1941.

Two cases are recorded in which metastatic lesions of an erysipeloid character—so-called carcinoma erysipelatodes—were mistakenly diagnosed.

In the first patient a radical mastectomy was done for carcinoma of the right breast (Grade 3) with axillary metastases. She received preoperative roentgen therapy with radium postoperatively. Five months after operation the wound had healed except for a small draining sinus at the apex, surrounded by an area of redness which was successively attributed to inflammation, post-radiation eczema, ringworm, a possible fungus infection, and self medication. As the lesion progressed, however, to involve the opposite breast, the back, and the abdominal wall, the condition was recognized as metastatic involvement of the skin, a diagnosis which was confirmed by biopsy.

The second patient had a duct carcinoma of the breast with metastatic deposits in the axillary nodes. Radical mastectomy was done following a course of roentgen therapy. The operative wound healed well except for a small area about which a pronounced erythema developed. This was at first believed to be due to local applications of azochloramide; later it was regarded as a radiation reaction, though the patient had received no irradiation postoperatively and any skin that might have been pigmented from the preoperative therapy had been removed. Five months after operation the condition was recognized as carcinoma erysipelatodes.

As each of these cases was at some time during its course mistaken for a radiodermatitis the medicolegal significance of the lesion is clear, and the importance of immediate biopsy is obvious.

Serial sectioning of biopsy specimens from the recorded cases suggests that the process is one of permeation of the subepidermal lymphatics and tissue spaces rather than of the venous channels.